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PHYSIOLOGIC PRINCIPLES IN THE MANAGEMENT OF DERMATITIS*

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PHILADELPHIA

THE greatest single source of disability from skin diseases is the large group of acute and chronic dermatitic eruptions. These diseases, in spite of having certain basic features in common, may vary widely in appearance, and a great number of terms based on morphologic differences or etiologic factors have been applied to them. This multiplicity of terms has led to much confusion. A few of these terms are sufficiently informative and useful to justify their retention in medical nomenclature. In the case of many terms applied to dermatitic processes, however, the words are so complex or meaningless as to indicate consigning them to obsolescence. It is proposed in this paper to discuss briefly the general principles that are of value in the interpretation of any case of dermatitis, and that have been found useful in treatment.

The device of "factorial analysis," a term proposed by Stokes,[1] is one of great value in the management of a patient with a chronic inflammatory eruption of the skin. In the use of this method, it is necessary to be constantly aware that chronic inflammatory skin diseases that have a single cause are rare indeed. As an example, the finding of fungi in an inflammatory eruption of the feet is often mistakenly regarded as having settled the issue. In most instances, the finding of fungi may be only an incidental one in the unraveling of the complex factors ordinarily operative in any chronic fungous infection of the feet.

The human skin occupies a peculiar position among the important organs of the body. The widely varying changes occurring in it are usually immediately apparent to the patient, his friends and his physician. This is both an advantage and a disadvantage. The lesions constitute an extraordinarily persuasive invitation to the patient to treat himself, and to change treatments frequently. As physicians, we are prone to employ a staggering number of complex chemicals in the treatment of the diseased human skin. These chemicals frequently interfere with the return of the tissues to normality, and often cause the skin to react in violent allergic distaste.

Under the heading of "dermatitis" are included eruptions that are characterized by varying degrees of erythema, some edema of the skin, frank vesiculation or oozing, and itching. In the healing or chronic phases of a dermatitis, the evolutionary changes, such as scales, crusts, lichenification and pigmentation, may be more obvious. Given these findings, it is convenient to classify the dermatitis as acute or chronic, to indicate whether or not it is of a specific morphologic type and to indicate the cause or causes if determinable. There is no need to introduce any of the more obscure types of "eczema" to confuse the issue.

Certain types of dermatitis have definite morphologic characteristics; their proper classification may be of great practical value. The most useful terms are: contact dermatitis, seborrheic dermatitis, atopic dermatitis, localized neurodermatitis, dermatitis due to ingested or inhaled substances, stasis dermatitis and mycotic or bacterial dermatitis.

These terms are sufficient for most purposes. They are certainly adequate as a framework for initial classification of any dermatitis, but they represent only the first of the steps necessary to evaluation of the eruption.

Causative Factors in Dermatitis

The following factors are of importance in the causation or perpetuation of a dermatitis:

Chemical irritation. It is a truism that the application of an irritant substance to an irritated tissue will ordinarily have only one result: more irritation. It seems worthy of mention, nevertheless, because observation of a large number of patients with skin diseases will yield many examples of violation of this basic principle.

The production of epidermal sensitization by the application of various chemicals in treatment. This is probably the single most important source of

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present-day dermatologic disability. Methods of inducing such sensitivity have reached full flower during the past decade. The cycle began with the demonstration that sulfonamides applied externally had a curative effect upon superficial pyogenic infections of the skin. Before the true sensitizing faculty of sulfonamides on the epidermis was demonstrated, ointments and other preparations containing sulfonamides gained wide popularity. There can be no question that the harm done by the external application of sulfonamides outweighs any possible good; the external use of any of these compounds at the present time cannot be justified.

The same sequence of events occurred with penicillin and to a lesser extent with streptomycin. Both these materials are strong sensitizers when applied externally, and should not be used in this way.

The external use of antihistaminic compounds has also attained wide popularity, although the true effectiveness of these substances in reducing itching or inflammation when *applied externally* has to date *not been demonstrated on the basis of controlled clinical experiments*. In controlled experiments in our department, Perry[2] and Baldridge[3] were unable to demonstrate any antipruritic or anti-inflammatory activity of Benadryl ointment or Thephorin ointment that might be attributed to the antihistaminic component rather than to the base. Benadryl seems fairly innocuous when applied to the skin, but Thephorin is unquestionably a potent epidermal sensitizer. We have discontinued its use entirely, on the grounds of uncertain clinical efficacy and undoubted harmful potentialities. Judgment must be reserved on the value of ointments or other preparations containing an antihistaminic compound for the treatment of any type of dermatitis, on the basis of the evidence available to date.

Still another group of potential sensitizers has come into increased prominence. These are the various "caine" compounds. Two of them, benzocaine and nupercaine, have had wide clinical use for many years. Occasional reactions to the former have been observed, but it is probably not a strong sensitizer. Nupercaine is capable of producing severe contact dermatitis at times, but its exact "sensitization index" has not been definitely established. Certain other compounds, such as Surfocaine, have recently come into wide use, and others will unquestionably follow.

It may well be argued that almost any type of medical therapy involves some risk and that frequently this risk is one worth taking. In the case of antipruritics, for instance, if a substance is shown to have a marked antipruritic effect, with resultant lessened scratching and secondary infection of an inflamed area of the skin, slight risk of sensitization might be worth assuming. However, on the basis of an extended survey by Shelley and his coworkers,[4] it appears that the clinical efficacy of these various local anesthetics has *in no instance been proved on the basis of controlled experiments*. In laboratory studies, these investigators have shown that the penetration of these agents through the unbroken skin in amounts sufficient to have any local anesthetic effect is virtually impossible; the skin is too effective a barrier in this regard. In the treatment of dermatitis, where some disturbance of the integrity of the skin has occurred, the purported antipruritic effects of some fifty-seven varieties of compounds have been recorded. In only two instances[5, 6] did the clinical experiment approach conditions of "planned design"[7] that would allow any conclusion concerning the real effectiveness of the "antipruritic" substance. That relief from itching does occur in conjunction with many methods of treatment cannot be doubted. However, the factors incident to such relief are many, and the psychosomatic aspects are profound. If an antipruritic agent has sensitizing properties, it is not too much to require that its effect on itching be unquestionably greater than that of many bland ointment bases. Such convincing evidence is not available on many compounds in common use.

Bacterial invasion of the dermatitic skin. This is a factor of the greatest importance, yet the role that bacteria may play in producing chronicity of a particular case of dermatitis is often overlooked. Data on this have been summarized in a previous paper.[8] The chief principles involved are as follows:

When the integrity of the skin is disturbed by dermatitis, whether of internal or external causation, its antibacterial powers against pathogenic organisms are greatly reduced. Within a few days to a few weeks, the bacterial flora changes markedly. These changes are: recovery of coagulase-positive hemolytic *Staphylococcus aureus* two to three times as frequently as from the normal skin and recovery of beta-hemolytic streptococci at least twice as frequently as from the normal skin, including the isolation of Lancefield Group A streptococci in some 15 to 20 per cent of cases. This organism is almost never found on the normal human skin other than as a passing contaminant.

This change in the bacterial flora of dermatitic skin may be evidenced by overt signs of an acute infection, including frank pustules, cellulitis, lymphangitis and lymphadenopathy, but frequently is not. The pathogenic or potentially pathogenic organisms may simply become apparently law-abiding members of the bacterial household, producing damage to the skin in insidious fashions that are not yet completely understood.

After widely varying periods of time, many persons with chronic dermatitis become sensitized to these abnormal bacteria, and particularly to staphylococci. Without any evidence of infection in the usual sense, the bacteria continue to produce toxins that may produce sensitization reactions. This sensitivity may at times be easily demonstrable,

but in some patients is not. The bacteria may be difficult to dislodge as members of the resident population and present methods of reducing the sensitivity of the skin to them (vaccines and toxoids) are unsatisfactory.

From this sequence of events, it may be seen how inevitable is some degree of bacterial infection of a dermatitic skin and how important is the prevention and treatment of it. In prevention, the most important method is to assist the skin in rapid regaining of normality, principally by not harming it. In treatment, penicillin, injected intramuscularly, is by all odds the least toxic and most effective method of dealing with such infection, although other antibacterial agents may be required in some instances. In spite of the apparent superficiality of the infection, parenteral administration of the antibacterial agent is probably superior to any type of local application.

General "broadening of the allergic base." The specific sensitivities of the human skin, whether epidermal or dermal, are sometimes remarkably constant, but at times highly variable. Some persons — "atopics" — are born with a capacity for sensitization of various tissues to a wide variety of inhaled and ingested materials. Some seem to have great sensitivity to external contactants, but certain others can never be sensitized to a material, such as rhus extract, no matter how frequently they are exposed to it.[9]

The following general principles are useful:

A patient who has become well sensitized to one contactant is ordinarily a fit candidate for sensitization to other substances. His range of sensitivities is therefore easily "broadened," and the greatest care should be taken not to expose him unnecessarily to such agents.

In the midst of severe exacerbations of a dermatitis, the range of contact sensitivities may suddenly increase markedly although, as a rule, temporarily. In such patients the application of relatively innocuous local medicaments, often substances that the patient has used previously with impunity, may produce reactions.

It has been definitely shown that patients who have developed an epidermal sensitivity through repeated contact with some type of medication may later develop a dermatitis when the causative compound is administered parenterally. This has been noted in thousands of patients in the instance of sulfonamides. It has also been observed frequently in patients treated with penicillin ointment. There is in many of these patients, therefore, an induced sensitivity in the dermis as well as in the epidermis.

It seems probable, although it is often difficult to prove in the individual patient, that areas of skin that have been sensitized in one way or another to a particular compound, or in which chronic secondary infection has developed, may develop sensitivity to certain foods.[10]

Patch and intradermal tests should not be employed during acute exacerbations of a dermatitis. The latter have a very limited usefulness in any event; their value is much overrated, in my opinion, and does not approach their usefulness in allergic affections of the respiratory tree, for instance.

Psychosomatic factors. It is obviously a mistake to regard psychic or functional factors as merely superimposed on a somatic disturbance; all chronic diseases of man inevitably have some psychic or functional component, but fortunately this component is not always the controlling factor. In the general group of dermatitis, the chief mechanisms involved appear to be related to itching (with development of a scratch–itch–scratch cycle), vasomotor changes and hyperhidrosis of the hands and feet related to tension states. This entire aspect of dermatology needs much more study; it is probably an important factor in almost every case of chronic dermatitis.

Functional vasomotor changes and organic peripheral arterial and venous diseases are of great importance in the perpetuation of chronic dermatitis. The patient with lividity and hyperhidrosis of the hands and feet is a perennial candidate for dermatologic disability. Whenever a mycotic or bacterial infection or a dermatitis of any character becomes well established on the hands and feet of these persons, it is extremely difficult to cure.

Persons with even moderate degrees of stasis dermatitis are subject to much risk of dermatologic disability, and not infrequently develop a generalized explosive "id" eruption, which may be extraordinarily persistent. Naide[11] has pointed out that the lower legs of these patients are extremely susceptible to sensitization from applied medication; this is in accord with our experience. We have observed several examples of generalized exfoliated dermatitis that followed the continued application of Furacin ointment to stasis ulcers or dermatitis, particularly when the compound was applied under a gelatin boot.

Mechanical blockage of sweat ducts as a factor in dermatitis. The means by which disturbances of sweating may produce diseases of the skin have been the object of speculation for many years. It was only during the recent war, however, that Sulzberger, Zimmerman and Emerson[12] and O'Brien[13] demonstrated independently that miliaria was associated with localized anhidrosis in the affected areas. This was an observation of practical importance.

Subsequently, Shelley and others[14] were able to produce miliaria experimentally. A short summary of these studies may be of interest: It was demonstrated that a wide variety of minor superficial injuries to the surface of the skin regularly produced

blockage of the sweat ducts. These injuries included moderate exposures to ultraviolet light, patch tests with soap solution and the application of aluminum chloride solution, adhesive tape and wet compresses for 48-hour periods. After a latent period of one to three days following the minor superficial injury, it was found that the areas treated did not sweat when the subject was placed in a hot-box. Superficial sweat-retention vesicles were demonstrable on the surface of the skin, without evidence of inflammation. It may be stated unequivocally that in a person who has had adhesive tape applied to his skin for 48 hours the area of application will not sweat for a period of at least two to four weeks. The same phenomenon will occur if a compress of ordinary tap water is kept in place for 48 hours.

A skin so treated will ordinarily return to normality with no sign other than slight desquamation. However, in susceptible persons who are kept in a warm environment, typical miliaria may develop from this treatment; this is unquestionably one of the mechanisms that contribute to dermatitis. The possible relation of this phenomenon to irritative methods of treatment is obvious.

There is good evidence that localized anhidrosis due to obstruction of the sweat ducts is present in atopic dermatitis, contact dermatitis, fungous infections, ichthyosis, seborrheic dermatitis and a wide variety of other skin changes. In certain cases, this interference with sweating may play no role in the subjective symptoms or objective changes; in other cases, however — particularly in a warm environment — the blocking of the sweat ducts may be a major factor in the production of itching, marked inflammatory changes and "tropical asthenia." Superficial fungous infections of the skin produce localized anhidrosis that may lead to the clinical picture of miliaria.[15] This is unquestionably an important mechanism in the development of acute inflammatory changes of the feet in many patients who have low-grade fungous infections, although the sudden recurrence is commonly regarded as due to growth of previously "dormant" fungi. This latter theory is inadequate to explain acute vesicular and bullous disease of the skin that appears within a few hours after increased sweating of the feet has occurred.

The factor of ichthyosis (the "dry-skin factor" of Stokes). The hygienic customs and artificially regulated environments of modern civilization place a severe strain on a person whose skin is dry, that is, lacking in sebum and other essential lipid components of the skin surface. More logical and effective methods of combating ichthyosis must await better knowledge of the physiologic changes involved. However, certain gross changes are clinically striking but are frequently not considered in the management of a chronic dermatitis, especially in patients in whom the gross classical features of ichthyosis are not present.

The recognition of a dry-skin factor in any dermatitis is important because on the basis of it one can ordinarily predict intolerance to certain types of medication. The prolonged use of drying shake lotions will almost always produce increased inflammatory changes in the skin. It is often necessary to use preparations of the emulsion type, or an ointment. Intolerance to soap is a striking feature. The use of various defatting agents such as hot water and synthetic washing compounds, lotions containing alcohol and preparations containing sulfur will promptly produce damage, as a rule.

In this type of skin, the use of soap substitutes probably finds its greatest field of usefulness. Frequent warm baths of any sort serve only to increase the inflammatory changes. The local treatment must emphasize greases rather than lotions.

The seborrheic or oily-skin factor. Chronic dermatitis in a person with an oily seborrheic skin is usually characterized by clinical features and courses that are quite different from those of a person who has a dry skin. There is often a more marked localization of the dermatitis in the so-called "seborrheic areas" — the scalp, retroauricular region, external auditory canal, folds of the nose and presternal and interscapular areas. More extensive involvement tends to be intertriginous. Secondary infection, particularly bacterial, is a marked feature, and the responsible organisms may be extremely difficult to dislodge from the resident bacterial flora of the skin. Cleansing of the skin and removal of epithelial debris is an essential part of the treatment. The most useful single local medicament is sulfur in one form or another, which acts through mechanisms that have yet to be determined.

Physical allergy, particularly photosensitivity. This very large subject, on which much has been written, can only be touched upon in this discussion. It is probable that instances of sensitivity to sunlight are far more frequent than is ordinarily suspected. The changes in the skin associated with photosensitivity may be extraordinarily diverse. There may be diffuse dusky erythema, resembling that seen in acute disseminated lupus erythematosus. The changes may be almost indistinguishable from those of a contact dermatitis. At times the lesions may resemble those of erythema multiforme or may be indistinguishable clinically and histologically from chronic discoid lupus erythematosus.

X-ray therapy. In taking the histories of patients with chronic recurrent dermatitis, one cannot fail to be impressed with the gross and indefensible overtreatment with roentgen rays to which many such patients have been subjected. Although many papers have been written on superficial x-ray therapy for inflammatory eruptions of the skin, particularly dermatitis, and although such treatment is accepted as "standard" in present-day practice, the exact value of superficial x-ray therapy in derma-

titis has *not been established* on the basis of controlled clinical experiments. Since such treatment admittedly has great potentialities for harm, it is not too much to require that its potentialities for good be much more clearly defined. In a few controlled experiments, we have been unable to demonstrate any absolutely clear-cut lasting effects of x-ray therapy on dermatitis.

Our data do not permit the statement that x-ray therapy of itself is not occasionally helpful in the treatment of a dermatitis. However, it may most certainly be stated that such effects, if any, will be noted after two or three exposures of from 50 to 100 r each. The use of prolonged courses of x-ray therapy in any type of dermatitis must be condemned. It is never justifiable in contact dermatitis, in my opinion. Its use in chronic eruptions, such as atopic dermatitis, recurrent severe seborrheic dermatitis, or chronic fungous infections may be regarded with grave misgivings. If such patients believe, correctly or not, that they have been benefited by x-ray therapy, they are quite likely to seek such treatment here, there and everywhere, with disastrous results. There is no worse skin change than late x-ray atrophy; substitution of this change, even in mild degree, for a dermatitis, however bothersome, is medically unjustifiable.

Cortisone and ACTH both have striking effects in producing regression of chronic dermatitis of various types. As the mechanism of action of these compounds becomes better understood, considerable clarification of the precise physiologic changes occurring in dermatitis should ensue. From the practical standpoint of treatment, however, we have been impressed with the rapidity and severity with which dermatitic changes may recur after cessation of therapy. It appears that if the basic etiologic factors are still operative, Cortisone or ACTH therapy has no more than temporary curative effects, and that "maintenance" doses may be required for a longer period than is safe or practical.

Dermatitis, acute or chronic

The essentials of treatment and prevention of acute or chronic dermatitis may be listed as follows:

Detection of responsible contactants and ingestants, or both.
- Sometimes simple, as in rhus dermatitis.
- More often difficult; sometimes impossible, but well worth the effort.
- Careful history and observation far more valuable than patch or intradermal tests.
- *No* tests during acute phase.
- Suspect *multiple* etiologic factors in any chronic dermatitis.

Treatment

Avoid primary chemical or physical irritants on already irritated tissue.

Suspect sensitization from previous local therapy.

Avoid local application of:
- *All sulfonamides*
- *All antibiotics* except bacitracin and aureomycin (?)
- Nitrofurans
- All "caine" compounds
- Antihistaminic compounds (oral administration preferable).

Allow for rest. Protect irritated skin by inert powders, as in "shake" lotion, or by bandaging.

Avoid ointments in most cases of oozing dermatitis.

Apply nonirritating compresses or soaks, such as 1:8000 $KMNO_4$, Burow's solution, physiologic saline solution, milk — warm or cold, whichever is more soothing.

Control infection by gentle removal of pus and epithelial debris (penicillin given parenterally the most effective and safest specific measure).

In chronic infected dermatitis, base treatment on complete bacteriologic study, including determination of sensitivity of bacterial strains to various agents.

Look for evidence of intolerance to any local medication.

Make a "*spot*" *test* of any "*new*" *local medicament* 12 to 24 hours prior to widespread application.

Control itching by means of:
- Rest of affected parts and avoidance of irritation from clothing
- Menthol and camphor (generally preferable to phenol)

Cool environment and avoidance of vigorous exercise if possible
- Sedation (antihistamine having most sedative effect; barbiturates usually well tolerated)
- Occlusive dressing at times to prevent scratching (old sheeting preferable to surgical gauze)

Avoid injection of specific antigens (such as rhus) during acute phase (this is worthless and dangerous).

As healing progresses, with reduced evidence of inflammation and with crusting and scaling, use ointments, such as:

Petrolatum
Hydrophilic ointment containing 0.1 per cent menthol and 1 per cent camphor
3 per cent ichthammol in ung. zo. USP.

X-ray therapy of uncertain value; in dermatitis, rarely indicated. *Possibly* some effect on inflammatory process (though mechanism not well established).
Decidedly dangerous in excess — particularly in areas normally exposed to sunlight.

Hospital Study and Treatment

Experience in military medical practice during the war demonstrated the marked value of the study of patients with chronic dermatitis under hospital conditions, *before* the process became too greatly changed by many secondary factors. Avoidance of disability from chronic dermatitis may be effected by careful study under the controlled conditions of the hospital environment. Because patients with dermatitis are usually ambulatory and following their normal occupation, however reduced their efficiency, it is sometimes difficult for them to appreciate the need for careful study for one or two weeks in a hospital. Hospitalization of such patients may be expected to accomplish the following:

More accurate determination of the patient's general medical status.

Removal of the patient from many of the conditions of his home or occupational environment that might be causing the dermatitis or contributing significantly to it.

Careful observation of the day-to-day course of the dermatitis.

Detection of possible intolerance to treatment that may be unnoticed under conditions of outpatient therapy.

Rigid control of the environment and of ingested foods and drugs. In some hospitals, control of inhalants is possible.

Assurance, especially if trained nursing personnel are available, that treatment is being carried out in a manner most likely to produce good results and prevent harm.

In achieving the above, the following hospital regimen has proved useful:

Temporary discontinuance of all local medication used previously, with substitution of a simple "shake" lotion, or petrolatum.

Temporary discontinuance of all internal medication, except in the occasional patient to whom regular administration of a particular medication is absolutely essential.

A carefully detailed history, with repeated questioning at different sessions regarding the possible role of contactants, ingestants and inhalants.

Careful, detailed general physical examination.

In some patients, barring of visitors for a trial period of at least two days, in an effort to determine whether or not they are carrying allergens to which the patient may be sensitive or are productive of significant psychic trauma to the patient (particularly the parents of children with atopic dermatitis).

Keeping the patient in a room that is as "allergen-free" as possible. Use of a plastic-covered mattress and pillow, absence of woolen rugs and dust-collecting hangings and prohibition of contact with cosmetics, flowers and plants are easily achieved in the average hospital room.

An initial trial elimination diet (provided that there are no medical contraindications) of either sugar solution and tea only for 24 to 48 hours, or a "basic" diet that is wheat-free, milk-free and egg-free. The recognition of a food factor in an eruption is often impossible except under conditions of hospital control. It is rarely determinable in dermatitis on the basis of skin tests.

Initial "spot" application of any new local medication at least 24 hours before it is used extensively.

Careful bacteriologic or mycologic study of the skin, or both (the latter is not obtainable in most hospitals).

Routine laboratory studies. Some of these are obviously essential. The most helpful include a complete blood count and determination of sedimentation rate, serum protein and postprandial blood glucose. Extensive biochemical "surveys" of the patient frequently do not yield enough information to justify such expense and effort. If the chronic dermatitis is unusual or not easily classified, a punch biopsy should be done, if for no other purpose than to establish a base-line of reference for the future.

Summary

By the use of many methods currently employed in the treatment of dermatitis, the patient is as likely to be harmed as to be helped. Adherence to a few simple principles will do much to prevent the frequently induced change of a simple acute dermatitis to a complex chronic one.

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PULMONARY ADENOMATOSIS*

Report of Two Cases

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PULMONARY adenomatosis is a rare tumor of the lung characterized by the presence of tall columnar mucus-secreting cells lining the alveolar walls, without evidence of bronchial origin or of primary adenocarcinoma elsewhere. In the past few years there has been a marked increase in the number of reported cases, with two recent reviews of the literature[1, 2] and numerous pathological discussions.

A considerable difference of opinion exists concerning pulmonary adenomatosis, or alveolar-cell tumors of the human lung, including complete disbelief in the existence of this condition as a separate entity.[3] The question of the existence of a true alveolar epithelium has not been settled, although the recent literature shows a strong tendency toward acceptance of this fact. Many theories concerning the origin of the condition have been suggested, among which are the infectious, the irritative and the neoplastic. The marked similarity to "Jaagsiekte," a communicable pulmonary infection of sheep, has stimulated a long but fruitless search for a causative virus, while the ability of carcinogenic hydrocarbons to produce similar tumors in mice has raised the question of inhaled irritants as a possible cause. Both these theories might explain the multicentric appearance of lesions described in many cases. The neoplastic theory is most commonly accepted, but even here there is no agreement concerning whether both "benign" and "malignant" forms of the disease exist.

It is not the purpose of this paper to contribute to the controversy concerning the etiology and histogenesis of this disease, but to present two additional cases, one of which (that of J. H. W.) is of particular interest in regard to postoperative survival.

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CASE REPORTS

CASE 1. J. H. W., a 59-year-old sheet-metal worker, was transferred to this hospital on July 25, 1947, because of a suspected malignant pulmonary lesion. In December, 1945, one and a half years before entry here, he was admitted to another hospital with a history of increasing cough in the preceding two to three years, marked fatigue and one episode of hemoptysis. The patient was a heavy smoker. Although a chest x-ray film taken at that time showed changes similar to but less marked than those described later, he was discharged without complete study or definitive treatment.

In May, 1947, two months before being transferred to this hospital, he was readmitted to the other institution because of persistent cough for four years, productive of a half cupful of thick white sputum every morning, dull, constant pain across the anterior chest, aggravated by breathing, frequent night sweats and a weight loss of 18 pounds in about two years. He stated that his sputum was occasionally streaked with blood, while fatigue and dyspnea on exertion were also becoming more noticeable.

Physical examination revealed a well developed, well nourished, florid, elderly man. His temperature was 98°F., the pulse was 88, the respirations 18 and the blood pressure 120/60. The chest was increased in its anteroposterior diameter, with equal expansion and no deformities. There was slight dullness to percussion posteriorly at the left base, but no rales or rubs were heard. A Grade 4, high-pitched blowing diastolic murmur was heard; it was loudest in the second right interspace near the sternum. The liver and spleen were not palpable. The extremities were normal, with physiologic reflexes.

Examination of the blood revealed normal findings, with a sedimentation rate of 40 mm. in one hour. Urinalysis was negative. Six sputum examinations for acid-fast bacilli and three for tumor cells were negative. A tuberculin test (O.T. 1:10,000) was positive. The prothrombin time, nonprotein nitrogen and serum proteins were normal. The vital capacity was 3.7 liters. The serologic and spinal-fluid tests for syphilis were negative.

An x-ray film of the chest on May 16, 1947 (Fig. 1), showed the left diaphragm to be elevated posteriorly, with several circumscribed areas of infiltration measuring 1 to 2 cm. centimeters in diameter visible in the region of the lingula. Additional chest films, fluoroscopy and bronchography yielded no further information. Intravenous pyelography, barium enema and upper gastrointestinal series were not remarkable, except for the presence of a previously discovered deformed duodenal cap.

An electrocardiogram was abnormal, showing right bundle-branch block and an old posterior myocardial infarction.

The patient's hospital course during the period of observation was uneventful. In view of the roentgenologic findings a tentative diagnosis of pulmonary neoplasm was made. It

was believed that the aortic diastolic murmur was rheumatic in origin, but in view of the possibility of syphilis in spite of negative serologic and spinal-fluid tests, he was given 6,000,000 units of penicillin over a ten-day period.

On July 25, 1947, he was transferred to this hospital for treatment of his pulmonary lesion. Physical examination and laboratory data were much the same as recorded above. Bronchoscopy on August 19 was completely negative.

On August 28, 1947, a thoracotomy was performed by Dr. Emerson Drake, who found a soft circumscribed mass in

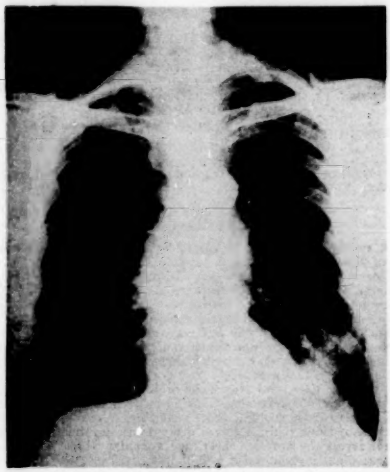

FIGURE 1. *Roentgenogram of the Chest in Case 1 (May, 1947), Showing Circumscribed Areas of Increased Density in the Region of the Lingula.*

the tip of the lingula, attached to the pericardium by inflammatory adhesions. These were divided and a segmental resection was carried out. No hilar nodes were palpated. The upper lobe contained a number of large bullous pneumatoceles, and there were many emphysematous blebs in both lobes. A frozen section suggested pulmonary adenomatosis; as the patient did not seem to be a suitable candidate for removal of the entire lobe, no further resection was done.

The pathological examination revealed a strip of anteroinferior portion of left lower lobe, including lingula, measuring 8.5 by 4.5 by 4.0 cm. and weighing 45 gm. The proximal (parenchymal) surface appeared normal, but on cross section the central third of the specimen was found to contain a nearly solid mass of gray-white tumor-like tissue, soft and gelatinous at the center. This mass extended to within 1 mm. of the lower part of the margin of resection. Microscopical examination (Fig. 2) revealed well differentiated high columnar cells, distended with mucus and having basal nuclei, lining the alveoli. The latter were filled with mucus and in places contained many macrophages. No mitoses or invasion of the subpleural or perivascular lymphatics was found; a thin margin of healthy lung tissue was present. The findings were consistent with pulmonary adenomatosis.

Postoperatively, the patient's course was entirely uneventful. He was discharged on September 18. He has continued in his usual state of health up to the time of this report, two and a half years later. At our request, he was admitted to the hospital in December, 1949, for a thorough follow-up examination. Although still troubled by a chronic cough, dyspnea on exertion and easy fatigue, he has maintained his weight, his vital capacity is unchanged, sputum examination is negative for tumor cells and there is no change in the appearance of his chest film (Fig. 3), despite the narrow margin of normal lung left at the time of operation.

CASE 2. *First admission.* J. E. P., a 56-year-old retired mounted policeman, was admitted to this hospital for the first time on October 6, 1947, with a chief complaint of cough and weight loss of 21 months' duration. In January, 1946, he first noted a mild cough and general fatigue. The cough eventually became productive of a cupful of thin mucoid sputum daily. He experienced occasional episodes of chills and fever, with gradual loss of weight and increasing

FIGURE 2. *Case 1. Area from Central Gelatinous Portion of Tumor.*

dyspnea. At no time did he have any hemoptysis. His use of tobacco was moderate.

In August, 1946, he was admitted to another hospital for a six-week period with the foregoing complaints. After complete studies, including two bronchoscopies and a bronchogram, he was discharged with a diagnosis of chronic pneumonitis of the right middle lobe. He worked for eight months after this, with persistent cough, dyspnea and weight loss, finally being forced to retire from the police force in May, 1947. He was admitted here five months later because of gradual progression of his symptoms.

Physical examination revealed a well developed, emaciated middle-aged man, who was coughing and quite short of breath. His temperature was 98°F.; his pulse was 74, his respirations 16 and his blood pressure 120/70. His chest expansion was equal bilaterally, but the trachea was deviated

to the right. There was dullness to percussion over the entire right lower chest, with bronchophony present over this area. The left chest was hyperresonant; no area of cardiac dullness could be made out. The heart sounds were loudest over the right anterior chest.

Laboratory data revealed a normal hemogram, with a sedimentation rate of 40 mm. in one hour. Urinalysis was negative. Repeated examinations of the sputum for fungi and acid-fast bacilli, including guinea-pig inoculation, were negative. Cytologic examinations of the sputum on several occasions were also negative. The serum protein was normal and a blood Kahn test negative. The vital capacity was 2½ liters.

An x-ray film of the chest taken on October 7, 1947, showed the right side to be markedly narrowed, with the heart and mediastinum displaced to the right. The right lung was poorly visualized, being obscured by the mediastinal shift, generalized increased density and atelectasis. Mottled infiltration was present in all parts of the right lung that could be seen, and a similar process was evident throughout the left lung, with the exception of the apex. On November 6, a fluid level was present in the right hemithorax; a small pneumothorax could be seen, confined to the apical and infraclavicular region. By November 26, the fluid level and pneumothorax had disappeared. On March 2, 1948, no appreciable change was apparent in the chest film, but on March 26 an almost complete pneumothorax was present on the right, with adhesive bands visible between the collapsed lung and the lateral chest wall. By April 10, there had been some re-expansion, but the intrinsic findings remained unchanged. A barium-enema examination and upper-gastrointestinal series were negative.

An electrocardiogram was normal.

The patient's hospital course was characterized by very gradual deterioration, showing varying degrees of dyspnea, cough and chest pain in conjunction with his changing chest picture as described under the x-ray findings. These consisted of bilateral diffuse pulmonary infiltrations, complicated by atelectasis on the right, mediastinal shift and episodes of hydrothorax and pneumothorax also on the right. He remained afebrile throughout his six-month stay.

Bronchoscopy on October 17, 1947, showed displacement of the trachea and carina to the right without evidence of endobronchial disease.

It was believed that the patient had a malignant lesion with diffuse lymphatic spread, but a definite diagnosis could not be made. He was given a therapeutic trial with a course of nitrogen mustard, and later a course of streptomycin in view of the possibility of tuberculosis, but neither of these measures had any effect. His weight declined from 122 to 117 pounds while he was in the hospital. He was discharged, unimproved, on April 18, 1948.

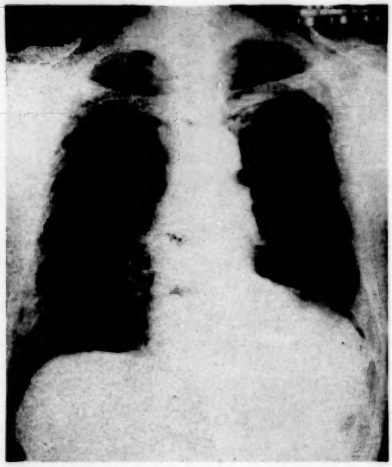

FIGURE 3. *Roentgenogram of the Chest in Case 1 (December, 1949).*
Two and a half years after operation, there is no evidence of recurrent tumor. Note the elevated leaf of the left diaphragm.

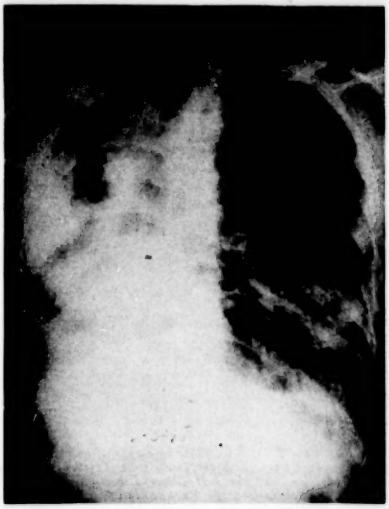

FIGURE 4. *Case 2. June, 1948.*
Note the marked mediastinal shift, bilateral pulmonary infiltration and atelectasis on the right.

Second admission. On June 2, 1948, six weeks after discharge, the patient was readmitted because of rapid progression of weight loss, cough productive of large amounts of watery sputum, dyspnea and chest pain.

Physical examination revealed him to be emaciated, cyanotic and in considerable respiratory distress, but the vital signs and chest findings were unchanged.

The hematologic and urinary findings were generally the same as on the previous admission. A blood culture was sterile. Further sputum examinations for acid-fast bacilli and tumor cells were negative. Liver-function tests were normal. A Congo-red test showed 71 per cent retention in the serum at the end of one hour. Arterial and venous oxygen saturation studies were normal. The vital capacity was unchanged at 2½ liters. The circulation time from arm to tongue (Decholin) was 18 seconds and the venous pressure was 110 mm. of water.

An x-ray film of the chest taken on June 3 (Fig. 4) showed a generalized haziness over the right chest, with further shift

of the heart and mediastinum to the right. On June 25, on the last film taken before death, complete collapse of the right lung, with a large pneumothorax, was noted.

An electrocardiogram was normal on admission, but on June 17, ten days before death, widespread ST-segment depression and lowering to inversion of all T waves appeared, the cause of which was not apparent.

During this second and last hospitalization, the patient remained practically afebrile but showed increasing dyspnea and cyanosis. There were several acute episodes of severe shortness of breath, accompanied by a shock-like state co-

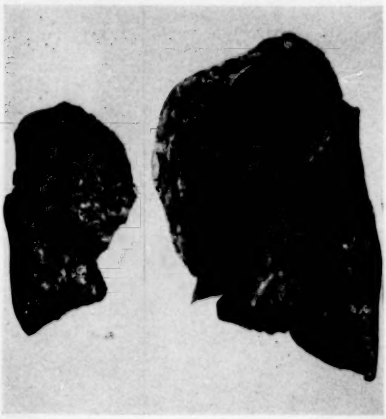

FIGURE 5. *Case 2. Cut Surfaces of Lungs after Frontal Section, Showing Collapsed Right Lung with Thickened Pleura, and an Incision into the Left Lower Lobe, which Presented the Gross Appearance of Pneumonia-like Consolidation. The apical bullae are collapsed.*

incident with recurrences of the previously noted pneumothoraces, only partially relieved by withdrawal of the air. Administration of oxygen and other supportive measures were of little avail. He required more and more sedation for relief of severe pleuritic pain. He died on June 26, 1948, two and a half years after the onset of symptoms.

Significant autopsy findings were limited to the thorax. The right lung was collapsed, measuring only 14 by 10.5 by 6.5 cm., whereas the left was voluminous (Fig. 5). At each apex were several air-filled bullae, the largest about 2 cm. in diameter. Fibrous adhesions were present behind both upper lobes. There was 175 cc. of hydrothorax on the right and 600 cc. on the left.

The right lung weighed 460 gm.; it felt meaty and noncrepitant, except for a softer area in the upper third of the upper lobe. It was encased in white thickened pleura. The cut surfaces were black, with firm-walled bronchi crowded together.

The left lung weighed 1540 gm., the pleura over the lower half of the upper lobe being diffusely thickened. The entire lower lobe and lower half of the upper felt consolidated, the firm moist pinkish-gray cut surfaces resembling those of lobar pneumonia and yielding turbid pinkish-gray fluid resembling purulent exudate. The bronchi showed no evidence of tumor, and the subcarinal and peribronchial nodes were moist and gray-black; they were not enlarged. In the pulmonary arteries were seen a few small well defined spots of atheroma.

The heart weighed 310 gm., with a right ventricle 5 mm. thick. Both ventricles appeared slightly dilated. The valves and coronary arteries were negative (as were also the spleen and liver).

Histologically, the alveoli in both lungs were lined with columnar epithelium and often contained much mucus. The alveolar architecture was distorted by interstitial fibrosis (increasing toward the pleura), with small pinched-off epithelium-filled spaces producing a false appearance of adenocarcinoma in places. Sparse or slight focal lymphocytic infiltration was present. The epithelial growth did not penetrate the vascular zone of the pleura (Fig. 6); it showed no mitoses, nuclear hyperchromatism or invasion of lymphatics.

The tumor cells in this case exhibited much less secretory activity than those in Case 1, but such a modification of

FIGURE 6. *Case 2. Section of Right Lung, Showing Interstitial Fibrosis and Failure of the Tumor to Penetrate the Vascular Zone of the Pleura, which is Greatly Thickened.*

functional activity can perhaps be accounted for by the degree of fibrosis and chronic inflammation present.

The principal pathological diagnosis was pulmonary adenomatosis.

DISCUSSION

Since Neubuerger and Geever[4] in 1942 collected 25 cases of pulmonary adenomatosis, another 27 have been gathered by Swan,[2] to which he added 9 more from the records of the Army Institute of Pathology. Ten cases[1, 5, 6-9] have appeared in the literature since Swan's review, which with the present 2 cases makes a total of 73.

It is of interest that only 4 of this total number have been subjected to operation, the diagnosis being made in all other instances at autopsy, although in one case[6] the diagnosis was strongly suspected on cytologic examination of the sputum and in another[10] on aspiration of the lung. As a matter of fact, if one holds to the multicentric theory of origin in pulmonary adenomatosis, the very advisability of surgical intervention may be questioned even when a definite diagnosis is made beforehand.

Of the 4 operations performed, 3 were lobectomies; one was a pneumonectomy. One of the patients in Swan's review[2] had his left lower lobe removed after discovery of a lesion by routine x-ray examination, and although he was alive at the time of reporting, there was radiologic evidence of disease in the right lung. The second patient subjected to lobectomy, described by Wood and Pierson,[11] died of uterine carcinoma six months after operation, while no follow-up data were given for the third case, reported by Drymalski, Thompson and Sweany.[9] The patient on whom pneumonectomy was performed, also included in Swan's review,[2] died six months later with a massive pleural effusion; no autopsy was performed.

It thus appears that the disease has been uniformly fatal among the reported cases, excluding those with inadequate follow-up or with clinical recurrences, which must be considered therapeutic failures. The first case in this report (J. H. W.) has survived for two and a half years after a segmental resection for pulmonary adenomatosis, without clinical or laboratory evidence of recurrence. This represents the longest survival time on record after a surgical attempt at cure, but it must be remembered that symptoms attributed to the disease are reported to have been of as much as eight years' duration.[12]

The great difficulty in making an early diagnosis is common in all malignant pulmonary conditions, made even more difficult by the routinely negative bronchoscopies in all cases and by the indeterminate results of cytologic examinations of the sputum in most instances. Whether or not extirpation of the lesion in the right middle lobe of our second case (J. E. P.) two years before his eventual death would have produced a cure is impossible to say, but in the light of the results of surgical intervention in the first case, there is a strong suggestion that the disease is not necessarily multicentric in origin and that early operation may offer a chance at cure, as in any other tumor of the lung.

Summary

Two cases of pulmonary adenomatosis are presented; the pathological findings are described.

One patient subjected to segmental pulmonary resection two and a half years ago is still alive, and shows no evidence of recurrence.

The value of surgical intervention in this condition is briefly discussed.

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CALCAREOUS TENDINITIS OF THE FLEXOR CARPI ULNARIS*

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BOSTON

THE relation between amorphous calcium deposits lying in close proximity to joints and acute attacks of pain in these joints has been recognized in recent years. Such deposits are most commonly found in the tendinous cuff around the shoulder joint. The symptoms of acute bursitis that these deposits may cause are well known. Similar acute attacks of pain may be produced by calcium deposits in the gluteal tendons, near their insertion into the greater trochanter. Less commonly seen are deposits in the capsules of the digital joints of the hands and feet, the triceps insertion at the olecranon and the extensor origin at the external epicondyle of the humerus or in the flexor and extensor tendons of the fingers. In this paper attention is called to the clinical picture produced by calcific deposits adjacent to or within the flexor carpi ulnaris at its insertion into the pisiform bone on the volar and ulnar surface of the wrist joint.

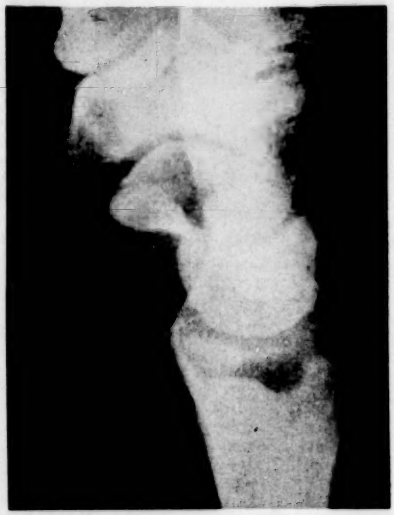

FIGURE 1. *Calcified Mass Probably Lying in Flexor Carpi Ulnaris Tendon.*

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Why calcium salts should become deposited in any particular region is not known. It is generally agreed that the primary lesion is probably a degenerative change within the tendon. As this pathologic change progresses, calcium phosphate and carbonate salts are deposited. The early deposits are microscopic and diffuse. These areas gradually enlarge and coalesce. In the early stages the calcium is in the form of crystalline powder and is found in multiple discrete pockets lying close together. In later stages these pockets merge as the intervening tendon walls degenerate, and the deposit assumes the consistence of cottage cheese. At the time acute symptoms are produced, the calcium is usually found to be contained in a single cavity, which is tightly encapsulated with the calcium either fluid or of the consistence of thin tooth paste.

The characteristic feature of all calcareous deposits is the development of acute symptoms. The onset of pain is often abrupt; within a few hours pain may become severe. Muscle spasm may be so marked as to prevent any joint motion. In the superficial joints, swelling, redness and local heat may become so marked as to suggest the diagnosis of either gout or acute cellulitis. In one of our cases lymphangiitis developed, apparently caused by the presence of irritating calcium salts that had become extruded into the soft tissues. In most instances, the acute pain is usually considered to be due to tension caused by acute swelling within the deposit.

Severe pain and swelling may remain for several days. Symptoms usually begin to subside within a week, and they disappear in two to four weeks. X-ray films taken at the time of onset usually show a calcified mass with well defined outlines (Fig. 1). Films taken two or three days later show the calcium to have become dispersed into the soft tissues. At the end of a month the deposit usually has disappeared.

Calcareous deposits within the flexor carpi ulnaris tendon present a clinical entity similar to the description above. The distinguishing feature is the sharply localized swelling, pain and tenderness directly over the pisiform bone. Five acute cases, and one chronic case, have been seen by the authors. The clinical course of the acute cases is shown by Case 1. None of the other cases, however, showed evidence of lymphangiitis.

CASE REPORTS

CASE 1. R. B., a 61-year-old laborer, developed very acute pain, with sudden onset, on the volar surface of the right wrist on February 14, 1948. The pain became so severe that he was forced to stop work on February 16. When he was seen on February 17, there was marked swelling over the

volar aspect of the wrist. The entire area was red and hot to touch. Extending up the volar aspect of the forearm were several fine red streaks of lymphangiitis. There was no epitrochlear or axillary-gland enlargement. The temperature was 99.2°F. and the white-cell count 9600. The patient felt well aside from the pain in the wrist. All wrist motions were limited because of pain. There was exquisite tenderness over the pisiform bone and x-ray films showed a large

FIGURE 2*A*. *Case 1. Lateral View, Showing Large Calcified Mass on Volar Surface of Wrist Joint.*

calcified area near it (Fig. 2*A*, 2*B*). The patient was hospitalized, and a posterior plaster shell was applied, with the wrist in slight flexion. Hot soaks and penicillin therapy were

FIGURE 2*B*. *Anteroposterior View, Showing Mass Lying between the Semilunar Bone, the Triquetram and the Ulna.*

instituted. Within twenty-four hours the acute symptoms had largely subsided. The temperature was normal, and the lymphangiitis had entirely cleared. At the end of five days the patient was asymptomatic and all signs, other than residual tenderness, had cleared. X-ray films taken March 1 (Fig. 3) showed the calcium to be widely dispersed in the soft tissues anterior to the wrist joint. The patient returned to work on March 8. It might be argued that this patient actually had a lymphangiitis that was cleared by means of penicillin. The complete disintegration and eventual absorption of the calcareous material indicate that this was the underlying pathologic condition.

Calcium deposits may also give rise to chronic symptoms, as demonstrated by Case 2. This case is of value, as operation proved the deposit to lie within the flexor carpi ulnaris.

CASE 2. C. M., a 36-year-old machine operator, sprained her left wrist on December 3, 1945, while operating a machine. Shortly thereafter, pain developed on the volar surface of the wrist. Within a few weeks, an area of swelling appeared over the flexor carpi ulnaris proximal to the pisiform bone. A diagnosis of ganglion had been made, but x-ray films showed a large calcified mass. On March 5, 1946, the area was explored through a transverse incision, under novocain. A definite bulge was found in the flexor carpi ulnaris tendon,

FIGURE 3. *Same Case, 12 Days Later. Only vague outline of original mass can now be seen in the lateral view.*

which on incision discharged a large amount of liquefied calcareous material. The patient had an uneventful convalescence and returned to work on April 8.

Acute calcareous tendinitis of the flexor carpi ulnaris follows the pattern of calcareous tendinitis as found elsewhere. The condition is most likely to be mistaken for an acute sprain or acute cellulitis. The symptoms come on suddenly and may become severe within a few hours; they are sharply localized over the volar aspect of the wrist joint, with exquisite tenderness over the pisiform bone. X-ray films showing the presence of a calcified area anterior and proximal to the pisiform bone confirm the diagnosis.

The acute symptoms rapidly subside upon immobilization and the application of heat. Surgical removal is not indicated in the acute stage but may be of value in the more unusual chronic lesion.

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THE DEEP SUBCUTANEOUS ADMINISTRATION OF CONCENTRATED HEPARIN

Observations on 15 Patients*

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HEPARIN and Dicumarol are at present widely used in the prevention and treatment of thromboembolic diseases. The former has maintained its place, despite its expense, because of its predictability, rapidity of anticoagulant effect and lack of side reactions. Since heparin is excreted or neutralized by the body in a few hours, it must be given continuously or intermittently over relatively short periods of time. Only the parenteral route has been found effective. The standard method has been the constant intravenous drip of 200 to 300 mg. in 1.5 liters of intravenous solution daily, as reported by Murray.[1, 2] Intermittent intravenous heparin, which involves less constant supervision and less inconvenience to the patient, is more frequently used[3-5] but is believed to produce a less continuous effect on the coagulation times. Heparin in a retarding menstruum for deep subcutaneous use has been adopted by many.[6-8] Because of longer action and inconstancy of effect this method has added problems of its own, one of which is the danger of longer action of a large fixed dose in a patient in whom the balance between adequate anticoagulant effect and serious hemorrhage may be delicate.

In our clinic, when wishing to take advantage of a rapid anticoagulant, we have adopted the use of concentrated heparin (100 mg. per cc.) injected intermittently into the deep subcutaneous tissue. Subcutaneous or intramuscular injection of heparin was tried by earlier investigators but abandoned because of unpredictable effects on the venous coagulation time and because of painful hematomas at the site of injection.[9] More recently it has been readopted by Neuhof and others,[10, 11] and painful hematomas have not followed the deep injection of a smaller deposit of the more concentrated product.

A group of 15 postoperative patients was selected for a careful appraisal of this method. The results are recorded below.

MATERIAL

Fifteen postoperative patients in whom it was desired to render the blood less coagulable for a short period of time were selected and given heparin according to the method described. The ages varied from 28 to 63 years; the lengths of time during which heparin was administered varied from two and a half to twelve and a half days. Coagulation times were determined by a modification of the Lee–White method. Three chemically clean tubes of 8-mm. diameter were used, each containing a glass bead that aided in the detection of the end point. The tubes were tilted each minute in succession after the blood in the preceding tube had clotted. The time at which the clotting blood in the last tube no longer allowed a bead to break through was considered the venous coagulation time. The blood of a group of 30 normal persons clotted in 5 to 8 minutes when this method was used. Blood was collected at the bedside after an accurate venipuncture of the antecubital vein. Admixture with tissue juice was carefully avoided, and the first 2 cc. drawn was discarded. Tubes were kept in a water bath at 37°C. during the determination of coagulation times.

Dosage of heparin was 50 mg. every four hours, given deeply subcutaneously over the deltoid or gluteal muscles with 2-inch 20-gauge needles. Coagulation times were determined immediately prior to such injections. These determinations were done four times a day for the first 9 patients studied and twice a day for the last 6 patients. When the coagulation time thus measured was over 40 minutes the dosage was reduced to 40 mg. every 4 hours. In the longer series this usually occurred after the second day. Lowering the heparin effect by increasing the time interval between injections to 5 or 6 hours resulted in return to nearly normal venous coagulation times before the next dose was given. A "priming" intravenous injection of 50 mg. of heparin given before starting the schedule increased the speed of heparinization but was not used routinely. Potentiation of heparin by Dicumarol, as reported by Walker and Rhoads,[12] was not studied. Caronamide was not used to decrease heparin excretion.[13]

RESULTS

The composite curves of venous coagulation times plotted against elapsed time are shown in Figures 1 and 2. A line is drawn through the means of the determinations. An arbitrarily desired range of venous coagulability between 15 and 50 minutes is indicated. In none of these patients did a dangerously high level occur during the course of medication. Seven of 225 recordings are slightly below the desired level. One patient developed a short episode of melena from his colostomy during

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treatment, but this did not recur although the same dosage was continued. No other clinical evidence of hemorrhage was observed. The injections were attended by pain only when they were too superficial. Occasionally there was a small lump at the injection site, but only one local hematoma (which was minor) was noticed. All injections were performed by nurses.

Six patients were followed at hourly intervals during the schedule; they showed only a 10- to 15-minute peak above the low points of the curve. This is unlike the sharp rise noted on intermittent intravenous heparin dosage, as shown in one patient in Figure 3. For further comparison, the coagulation times as followed on a patient receiving 2 400-mg. doses of heparin in a retarding menstruum are shown in Figure 4. Other similar results of the latter two methods are not included.

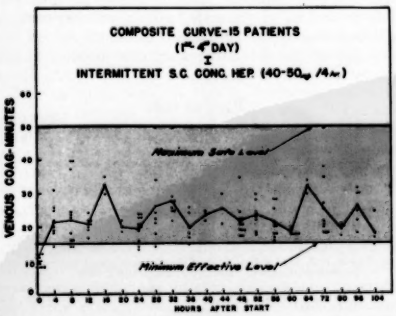

FIGURE 1.

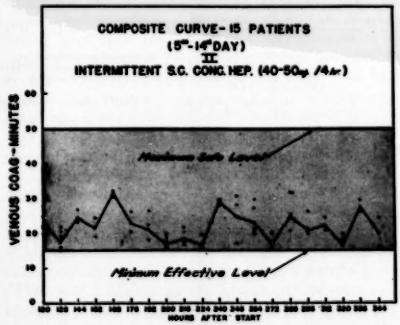

FIGURE 2.

Prothrombin times were determined in 6 patients after the method of Quick,[14] but no significant change was noticed during heparin medication four hours after a dose.

During the routine use of this heparin schedule on 35 other patients, venous coagulation times were determined only once daily. Protamine and toluidine blue, as antagonists to heparin effect, have not been needed on either this study group or the much larger group in which heparin was used for prevention or treatment of thromboembolism. No thromboembolic episodes occurred in either group while they were on the above schedule.

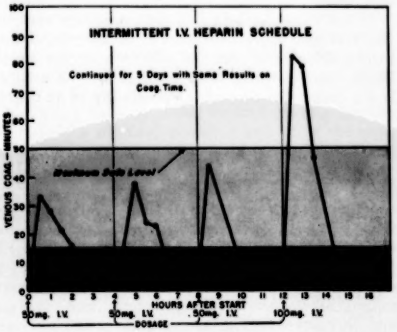

FIGURE 3.

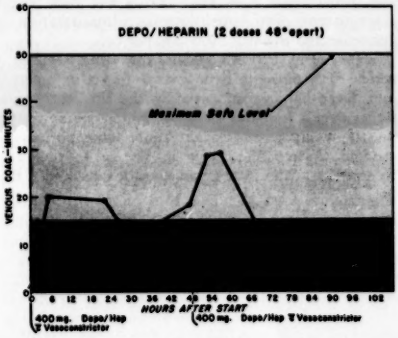

FIGURE 4.

DISCUSSION

Heparin is useful in altering the coagulation of blood. It is expensive, however, and must be given

parenterally. Constant intravenous drip is a cumbersome method, but it is still the standard method that others attempt to equal. We have found that the method described combines convenience with no increase in cost. Table 1 shows the approximate daily cost of the heparin required, excluding nursing and laboratory expenses, by the alternative methods described.

In our clinic heparin is most commonly used on the first two days of a course of anticoagulants, before Dicumarol can be effective, or for much longer courses where Dicumarol cannot be used or is not desired because of the possibility of an operation. Heparin effect is easily and quickly overcome, if necessary, by stopping administration of the drug and waiting a few hours, or by the administration of either protamine or toluidine blue.[15, 16] The latter two drugs are now readily available.

TABLE 1. *Daily Cost of Heparin per Patient (July 1950).*

METHOD	AMOUNT	DAILY COST FOR SMALL AMOUNTS*
Constant intravenous drip	200-300 mg.	$5.60- 8.40
Intermittent deep subcutaneous	240-300 mg.	6.72- 8.40
Intermittent intravenous	300-400 mg.	8.40-11.20
Depo-heparin	200-300 mg.	7.65-11.48
Heparin-Pitkin	200-300 mg.	5.00- 6.80

*Not gross quantity.

The variability of the effect of heparin in retarding menstruums now available is not met in the intermittent deep subcutaneous administration of concentrated heparin (100 mg. per cc.).

Heparin in Pitkin's menstruum was used in a series of 4 patients over a two- to six-day period and Depo-heparin was used in the three other patients over a four-day period, with inconstant effect on the venous coagulation time. One patient so studied showed the curve in Figure 4 previously referred to. The latter two methods showed a disappearance of heparin effect after eighteen to twenty-four hours with the 400-mg. dose, which could be compensated for by using 600 mg. or more on the first day. This increased the daily cost.

The use of subcutaneous heparin also has the advantages over the constant intravenous drip method of requiring less constant supervision and of allowing the patient to be semiambulant.

CONCLUSION

The intermittent deep subcutaneous injection of concentrated aqueous heparin (100 mg. per cc.) has proved in 15 patients to be a reliable method of obtaining hypocoagulability of blood.

Some of the advantages of this method of administration are discussed.

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FIFTY YEARS OF MEDICAL PROGRESS

MEDICINE AS A SOCIAL INSTRUMENT: NURSING

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TO ATTEMPT to discuss the progress in nursing during the past 50 years is to face a baffling task. It must be admitted at once that the story is one of confusion — great enough that it might be considered by some critics to exclude altogether the use of the word progress. But before we concede any such decision, much needs to be said, and much can be said.

Let us look, then, at this half-century, namely, the years between 1900 and the present. What has been happening, be it labelled progress or otherwise? What are the boundaries of this service called nursing? What does the word nursing indicate today? Why, as is obvious, has the quantity of service expanded so greatly during this time? Why, in spite of the phenomenal growth in numbers, is there always a shortage of service? Why is there so much discontent with the quality of nursing, and particularly with the quality as given in hospital? Why the extraordinary lack of reasoned argument concerning the conduct of nursing schools? Why are nurses themselves always on the defensive, and almost everyone else on the offensive in regard to nursing? Why must I myself dread the effort to discuss nursing with even my most intelligent friends, when I know that seldom will such discussion get past prejudices based upon ignorance of the real fundamentals of the argument? Why are these fundamentals so elusive? Why am I wondering now how many hospital administrators or medical practitioners — or even what number of nurses — will read this article patiently and with a desire to understand, or with the thought that some new light might appear? It will serve no purpose to open a discussion of nursing unless these questions are recognized at the outset. So much controversy cannot be ignored. Can we find a reasoned and convincing argument to explain the past and give promise for the future? This is my hope in this present writing.

My claim is that the key to an understanding of the whole situation is to be found in a careful study of the social history of the nineteenth century, and especially of the influences that pressed heavily upon the organization of nursing services in that period. If it is conceded that nursing is attempting to serve the mid-twentieth century while living in a strait-jacket fashioned nearly a hundred years ago, much of the fog of misunderstanding may be dispelled, and the way made clear for constructive action. Particularly should we try to discover why the nineteenth-century fetters are still fastened so firmly around nursing — as we believe they are — while other professional groups have escaped these fetters to a large extent.

Glancing back to 1850, we find nursing for the most part a despised domestic service in the relatively few secular hospitals of that period; and it should be noted that neither then, nor at any previous time, did nursing have any real connection with medicine. Apart from the work of the religious orders, where the protection afforded by the order itself maintained discipline and devotion in this service, we find that hospital nursing was an occupation for those women who could earn a living in no other way. The daughters of respectable families did not leave their homes for occupation of any kind unless forced to do so by poverty. There was no thought of even secondary education for girls, and no formal preparation to qualify young women for professional work. Certainly there seemed to be no content in nursing that required either education or training. Furthermore, there was little concern for the miserable condition of the sick poor in the lay hospitals. Finally, the practice of modern preventive medicine was yet to be born, as was also the presumptuous thought that all classes, rich and poor alike, had the same claim to health protection.

Thus came the second half of the nineteenth century: and then appeared in quick succession a trio of influences that have combined to produce the nursing situation of today with all its confused struggles. We see, first, the extraordinary development in the practice of medicine, curative and preventive; secondly, the developing status of women and new provision for their education; and thirdly, a growing sense of social responsibility that forced particular attention upon the social and economic aspects of sickness and health. These three streams of influence should be examined one by one.

First: the growth of medicine. What happened in this field in the nineteenth century that pushed nursing abruptly into an ever-widening range of activity? The reply is found in terse form in Sir George Newman's words, written some twenty-five years ago: ". . . In the last one hundred years men have witnessed a growth in the science and art of medicine incomparably greater than in any other similar period in the history of mankind" — a growth that has proceeded continuously since Newman wrote these words in 1924. By the end

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of the nineteenth century the application of this new medical knowledge was bringing much new content into hospital practice with, consequently, an ever-increasing demand for more nurses, and for nurses able to do more nursing of a kind and quality never before dreamed of. At the turn of the century this had started slowly enough, but soon it became a mushroom growth. At last nursing had become directly associated with medicine. Then suddenly came a further complication in the request for nurses to share in the rapid development of public-health practice, though this was not particularly noticeable until the second decade of this century.

Next, we note that the second half of the nineteenth century was bringing a radical change in the position of women as persons, and as active

FIGURE 1. *A Hospital Ward at the Turn of the Century (Courtesy of the Montreal General Hospital).*

members of society. By the end of the century, through the heroic efforts of devoted leaders, secondary schools and universities were being shared by both sexes. True, there was not yet equal opportunity in education for the woman, but long strides were being taken toward this. At the same time, women began to enter a number of the professions; indeed, the barrier was down from the time that Elizabeth Garrett Anderson qualified in medicine in 1865.

From these two influences — medical developments and the woman's movement — *the woman as nurse* has, in our own half-century, 1900–1950, been living through a period of rather frantic effort to adjust to a greatly changed social rôle as a woman and, at the same time, to adjust to a greatly changed social demand as a nurse. The contrary winds have made this a painful process.

Finally, we trace the effect upon nursing of the movement toward social reform that spread from the late eighteenth century and throughout the nineteenth — reform offered first by the privileged on behalf of the needy and later demanded by all classes on their own behalf. Influences were at work to fix attention upon one after another of the weakest groups in society: the prisoners, the mentally sick, unprotected children, the slaves of the new industrial conditions, and finally the sick poor, particularly the homeless and destitute in hospital. Leaders appeared who campaigned in each field in turn: John Howard, Pinel, Theodore Fliedner, Elizabeth Fry, Lord Shaftesbury, and among others, Florence Nightingale, the powerful but even yet little-understood woman described by Strachey as possessed with a demon. Strachey's description, though slightly sensational, throws more light upon events than the usual representation of the "ministering angel."

At Miss Nightingale's name we must pause and make an effort to assess her contribution to nursing. Other attempts at reform in this field had preceded hers for several years, but the efforts had turned back to old forms — especially the Deaconess Order — and had sought to revive these and make them serve again by adding new patches to the old garment. It took a woman of Florence Nightingale's genius to realize the need for radical change. Sir Edward Cook, her official biographer, says that she may be called "the founder of modern nursing because she made public opinion perceive, and act upon the perception, that nursing was an art and must be raised to the status of a trained profession."[1] What Cook has emphasized is that Miss Nightingale was the chief architect of an institution new in the history of the world: the nursing school. Certainly she was far in advance of her time when, before 1860, she realized that there was already enough content in nursing to demand formal preparation, and that this preparation should be given in a school organized for the purpose. She realized also that only in this formal fashion could a respectable secular service be created. Her work was brilliantly constructive.

Up to this time, the Gamp sisterhood had been the usual source of domestic care (!) for patients in the lay hospitals of both the old world and the new. The almost incredible stories of Bellevue's wards in 1872[2] provide a good illustration of the miserable conditions in many hospitals at that time. Even so, ridicule and criticism were poured upon Florence Nightingale when she sought to put an end to such conditions; there was the same spate of conflicting argument as is heard at present. The public conscience could soothe itself with remarks such as that quoted by Lord Granville: "Lady Pam . . . thinks the Nightingale Fund great humbug. The nurses are very good now; perhaps they do drink a little, but . . . poor people, it must be so tiresome sitting up all night, and if they do drink a little too much they are turned away and others got." In spite of this kind of irresponsible chatter, the Nightingale school took form in 1860 in con-

nection with St. Thomas's Hospital in London, and soon proved its value to hospital and patients alike.

The next step in the development of nursing — during the last three decades of the century — demands the most careful examination, for here seems to lie the cause of much of the trouble that has followed. Other hospitals sought reform and, gradually, attempts were made, in one place after another, to reproduce the pattern of the school established at St. Thomas's. All over the British Isles, in many countries of the British Empire, in the United States and in the northern countries of Europe, hospitals introduced what was called the Nightingale system of training nurses and of nursing patients. Unfortunately, from the beginning the copy was not exact. The Nightingale school had been organized as an independent institution, self-supporting and distinct from the hospital; apparently the pupils were meant to be supernumeraries, added — at such times and in such places as the school dictated — to a nursing staff already employed to give service to the hospital. But when copied the school lost its essential characteristic of independence and was turned into what is now called a hospital school of nursing — that is, a system of hospital-administered apprenticeship, the "historical accident" of Dr. Esther Brown's comment.[3] Apparently no one had noticed that it was the income from its endowment fund that had made the Nightingale school possible, by giving it independence, and therefore the authority to govern its own procedure. For the succeeding schools there was no endowment, and no other form of income. Lacking this, the school proper was lost: the students became apprentices who were required to work their passage through the training course and consequently were required to service the hospital. Worst accident of all, the Nightingale school itself imperceptibly but surely lost its original form and slipped into the mold of the inexact copy; in place of the distinct one-year training course, the school (?) was found a few years later to be offering three or four years of apprenticeship, and the patients were being nursed to a large extent by the service of pupil nurses. The vestigial remains of the original school exist today in the fact that all student nurses at St. Thomas's live in the residence called the Nightingale Home during their first year of training.

Strange as this story may appear, it is really not difficult to understand how it could happen in the last quarter of the nineteenth century. In the relatively simple conditions of hospital nursing in the 80's and the 90's, the difference between a formal school and an apprenticeship system could, and did, pass unnoticed; indeed, any form of training was bound to give satisfaction as compared with the former unorganized and sordid conditions. But the change from pupil status to apprenticeship gave rise to tragic consequences in a later, more complex period.

The real difficulty lies in the effort to explain the unshakable power that the apprenticeship system has wielded ever since. Unfortunately, by the end of the nineteenth century several influences had been at work to fix this persistent form, in spite of the changing world of both medical and educational practice in which it was expected to serve. Obviously the system had great economic value for the hospital, so hospital authorities welcomed it. Also, the nursing profession itself seemed to accept it without question. It does not appear that thought was given to the growth of educational procedure in other fields, or to the great change that was swiftly taking place in the practice of nursing itself — change that was soon to make the hospital training school more obsolete with every passing decade.

And so we have come to our own period, the first half of this present century. All that I have written so far has been offered as necessary introduction, and yet the introduction has projected the story of today at many points. Without this explanation from the past, the confusions of the present might be looked upon as a Mad Hatter's tea party, but, with the explanation, we see the inevitability of at least part of the present situation. In spite of all the contradictions and controversy, I am quite ready to claim that new foundations have been laid in this present century; it now remains for me to describe the progress that has been made in this time. This progress may be considered under four headings: in the content of nursing; in the personnel of nursing; in the conditions under which nursing is being done; and in the preparation for nursing, that is, in the field of nursing education.

First to be considered is the development in content. Passing by the self-directed ministrations of Mrs. Gamp and her friends, we see the picture of the arduous but simple work of the women of the earliest Nightingale era, including the probationers and the staff nurses of both the "old style" and "new style" of Henley's verses. By 1900 this content had settled quickly into much domestic work, fairly simple technical skills and even the appearance in district nursing of some social significance when the individual nurse sensed her opportunity to add this, although she had had little formal preparation for it. This great adventure of visiting nursing was forecast by the district nurses of William Rathbone and his contemporaries in England, and later was given vivid impetus in the 1890's by Lillian Wald and her associates in the United States and by the pioneer nurses of the Victorian Order in Canada and similar associations in other countries. It should also be said that from the end of the past century there were some hospital nurses of high caliber who recognized their opportunity to give their patients something more than technical service: new hope and strength, and

healing of mind and spirit as well as of body; while some — also untaught — were using their unique opportunity of contact with patients — much more prolonged than that of the physician — to stimulate or contribute to research. All interpretation of these words must be related to the period.

Today the content of nursing has expanded beyond all possible anticipation of a century ago. Even Florence Nightingale, with her great gift of imagination, could not have foreseen today's nurse, who is often called upon to perform with faultless precision more intricate and responsible treatments than those given by physicians when the Nightingale school was born; she could not have foreseen the great number and size and variety of today's hospitals, with thousands of nursing schools dotted all over the world, and the consequent demand for "lady" superintendents, supervisors, instructors and many, many specialists in nursing services. And, most interesting of all, Florence Nightingale, in spite of her understanding of the service that a nurse might possibly give as a "health missioner," could not have realized the breadth and depth of the health field in which many nurses are now asked to serve, a field where medicine and social work meet, and where the nurse must play the double rôle of medical and social worker.

FIGURE 2. *The Expert Nursing Committee of the World Health Organization, Geneva, 1950.*
Front: Miss L. M. Creelman (Canada), Miss E. W. Brackett (United States), Miss D. C. Bridges (England), Miss O. Baggallay (England). Rear: Mrs. L. Patterson (United States), Miss M. Dunillard (Switzerland), Miss Y. Hentsch (Switzerland), Miss F. N. Udell (England). (*Courtesy of* The Nursing Mirror, *London, from an official United Nations Photo.*)

In summing up the progress in the content of nursing during these 50 years, we see the growth from very small numbers to an enormous army; progress from limited technical service to a wide variety of services, many of professional quality; progress from ancillary status in the sickroom to partnership in the medical services; progress from hospital and sickroom duties alone to participation also in social work that touches the community at many vital points; progress from nursing as a matter of small and somewhat local concern to the 1950 organization of the Expert Nursing Committee of the World Health Organization. It is little wonder that this rapid growth has caused some confusion.

Secondly, we recognize progress in the type of young woman found in nursing. This is an interesting story, starting from the days of Victorian snobbery and continuing to our own time. Undoubtedly the early Nightingale era brought a sprinkling of recruits from homes of culture, but there is considerable evidence that general opinion, then and all through the intervening years, has tended to relegate nursing to those of mediocre intelligence and limited social background. There is much of this same thought today — especially in the secondary schools from which recruits must come. Often, intelligent and gifted students are directed firmly away from nursing when they show interest in this field. The struggle to bring about a favorable attitude toward recruitment is expressed clearly by two contemporary observers from the field of sociology in the following words: "A socially postulated 'natural' affinity between femininity and . . . 'drudgery' is . . . a near-universal phenomenon, and affected rather deeply (that is, in the nineteenth century) the definition of the role of the nurse. The most immediate historical source of this definition is the Graeco–Roman and (the) English gentleman's aversion to manual work. This orientation was clearly present also in Florence Nightingale's practice of dividing her trainees into 'lady probationers' and 'nurse probationers.' The latter group — recruited from among literate servants — was especially active in those parts of the hospital work that corresponded most closely to simple housekeeping duties. . . . The fact that, for several decades, most trained 'bedside' nurses working in England were recruited from amongst the 'nurse-probationers' rather than from the ranks of the genteel 'lady probationers,' probably also exercised an appreciable influence upon the social definition of the status and function of the nurse."[4] In spite of these indisputable facts, there has been progress in the selection of personnel for nursing. We make no extreme claims, for it is freely admitted that nursing has the same admixture of desirable and undesirable recruits that is found in all professions in today's democracy. Nevertheless, increasingly, young people with first-class ability are found in nursing schools (though not always remaining!); and increasingly, though certainly not always, these students represent the best homes that our countries have to offer in terms of education and culture (in the best sense of this

much abused word). Progressing from a decidedly poor social status, the nurse now receives a qualified recognition as a professional worker.

The third aspect of this progress is the material and physical conditions of the work of the nurse, a subject that receives much attention from the public at large. Important as it is, we wish that it would not always be mentioned first in public debate, or even appear to be the only matter for concern. Certain conditions have improved greatly, with the result that many nurses are now leading a much more normal life than has been possible at any other time since secular nursing was first organized. After long delay, this improvement has very recently taken place. I agree that it is important, and a matter for congratulation, that many nurses are working not more than eight hours a day, that many have one free day in seven, that some are receiving an income permitting a modestly comfortable way of life, with protection against unemployment, sickness and old age. It is significant that a few may even leave salaried positions in order to obtain added education to prepare for a special field of service. Another progressive step is found in the correction of mistaken thought on the part of nurses themselves. Earlier in the century we were narrowly exclusive, fearful for our status as "trained" nurses, and afraid of the effect of recognizing an assistant group called at that time "practical nurses" and known today by various names. Indeed, we blocked all such suggestion. Fortunately this attitude has changed, though there is still much progress to be made in establishing wise and happy relations. But these early fears are not going to be soon forgotten or forgiven by our critics; nursing must still live down the unfortunate reputation of having refused to share its services with an auxiliary group.

We are glad to stress all this progress in the conditions of nursing service, but we are bound to bring into the light one other condition, one in which it is not possible to record progress. This bears upon the hospital nurse's work. I refer to the devastating effect felt by good nurses (students and graduate staff alike) when they do not have time to nurse their patients properly. We are forced to conclude that this inability to give adequate time to actual nursing, and thus to afford satisfaction to the nurse herself and to her patients, is the main cause of the constant turnover in staff and consequent lack of stability in the nursing service of many hospitals. It even causes many young graduates to turn quickly away from the hospital as soon as their diplomas are secured. This feeling of frustration and the consequent instability in the service form a vicious circle, which will be broken only when those with power and authority recognize the situation and make a determined attack upon it. The recent world war increased this pressure in hospitals, but the condition was evident long before 1939. Those who have studied the problem most carefully are convinced that the root of the trouble lies in the organization of the nursing school in the hospital and the consequent confusion in a service shared in extraordinary proportions by students and employees. In recent years, the nursing profession itself has unceasingly called attention to the folly and danger of the situation. Year after year, proposals for reorganization have been offered, but with rare exceptions hospitals have been unwilling or unable to take action. A further word is appropriate enough here. Since possibly the nursing profession is now receiving its full quota of the young womanhood of some of our countries, the soundest economy should be practiced in the use of the time of these skilled practitioners.

Finally, in this fourfold recital of progress, comes the topic of nursing education. Paradoxically, our fifty-year span seems to have brought great change, and yet also to have maintained a remarkable sameness. The worst feature of recent years is that while the content of nursing has been developing with great rapidity there has been no corresponding *recognition* of this development. Those who have had much influence in the whole field of nursing education seem to be blind to the fact. Hence, it is folly to hope for logical action on their part. But why this lack of understanding concerning the scope of nursing service? My own conviction is that a curious fixity of thought is obscuring the interpretation of the word "nursing." Through centuries of usage, the word has become chained to a meaning so restricted that it cannot represent anything beyond bedside care; and by most people this bedside care is thought to consist only of domestic service for the patient, plus a few simple treatments, *always under the doctor's immediate direction.* With such a masterpiece of misrepresentation, what is to be done? Again I quote Dr. Esther Brown: surely this same thought was in her mind when she spoke of nursing as "a profession weary from the heavy weight of traditionalism."

I wish I had space to trace the story of the hospital nursing school during this half century, and to describe the sorties that have been made from this school into other institutions, in a somewhat frenzied effort to accomplish the impossible. It is an interesting tale, but there is no place for it here. Nurses themselves must accept responsibility for many mistakes during these years, for inconsistencies and for grasping often at the shadow rather than the real substance of reform. But the story reflects less credit upon many others who have been in a much better position to understand the educational resources of the community and the appropriate use of these. Hospital authorities, government officials and the senior profession of medicine — all these have been transferring new responsibilities to nursing in steadily increasing quantity ever since

the opening of the first world war, but these same groups have done little to support the development of nursing education in corresponding measure. Indeed, there has been considerable sustained neglect, and even definite opposition to progress. The old school-cum-service pattern persists, and is defended with extraordinary zeal. We are grateful to many individuals in these same groups who have given help, but some impression must now be made that will produce a general change of attitude. The present situation is shocking in many respects. Indeed, considering that our educational resources are so limited, some of the increasing demands upon nursing frighten us. The new civilian defence proposals may be in this category.

Certain changes must be made if the nurse's education is to keep pace with these demands. First, the idea must be accepted that all nursing schools should be divorced from hospital administration and re-established as educational institutions. In this case the hospital would have to content itself with its own legitimate responsibility: the employment of a staff to nurse its patients. With this arrangement, it should be easy to fit students into the nursing practice, but also to keep them always under the control of the school authority. As far back as 1923, this change was forecast in a remarkable document, popularly called the Goldmark Report,[5] that was published that year in the United States. Since then, further support for more or less radical change has come from various countries.

It is now quite obvious that schools of nursing of two distinct types are urgently needed. Both have taken form already, but they are very few in number, and even these are fighting for their lives. The one type, perhaps to be called the junior school, is — at its best — taking the form of an exceedingly good technical school that provides the technical training in an atmosphere of science and humanism. This atmosphere can be maintained only by an excellent teaching staff: that is, by nurses who themselves have had the best of technical training combined with broad general education in a university school of nursing. The junior school of which I am speaking may remain on the hospital grounds, as at present, but financially and administratively it should be independent of the hospital. Thus we can prepare the great army of clinical nurses now so necessary to the community's social and medical services.

Secondly, the resources for nursing education must include the university school. The number of nurses to be prepared there will be relatively small, but this school is nonetheless essential. At least three special groups need this educational opportunity: the executive staff for hospitals, the teaching staff for all nursing schools and the medical social workers known as public-health nurses. As there is still much need to defend university education for nursing, let me add some comment. Consider the great and growing number of hospitals. Does the community not understand that nursing provides a very large section of the professional service given in every one of these hospitals? Do we stop to realize that this service does not just happen? Do we not know that nurses must administer this service, and that they cannot administer wisely on the strength of technical skill alone? Only broadly educated citizens can handle today's problems in this vast network of institutions. Look at the woman who is asked to direct the nursing service, with all its complexities, for a hospital containing hundreds of patients whose lives and future happiness may be at stake. Do we see that this nurse-administrator has the opportunity to coordinate all the services so that the work of her professional colleagues may have greatest success? Do we see also that such a person can seriously disrupt this organization if she is inadequate? Is there any more appropriate task for a university than the preparation of this citizen, call her what you will? Consider also the teaching staff being prepared for the junior as well as the university schools of nursing. What teaching have these staff members to do? Just a bit more than the clinician in medicine of not so very long ago, and just as much as the instructor who is developing the professional philosophy of the students in any school of the university. Is there any doubt that some of the young recruits for nursing need general education on the university level?

We should be able to explain and justify what is needed in both content and method from the university school of nursing. The explanation is both simple and profound. The intention is to prepare professional women who, through study of the humanities and social sciences, will grow in understanding and wisdom; with this education in the realm of human values, they may approach with some degree of safety the work that is awaiting them. For these young people, we must "save general education and its values within a system where specialism is necessary."[6] And specialism is necessary, for these same students should have nothing short of excellence in their own technical skills and knowledge. Obviously, this preparation will include a sound scientific basis. We wish to combine the technical with the general subjects throughout the whole university course, believing that the richer result will be obtained by this method.

Amidst a welter of irregular and deplorable arrangements between hospitals and universities, a very few of these fully developed university schools of nursing have taken form. These are organized on the same basis as the other professional schools in the same universities, and offer the regular pattern of degree courses. But let me repeat that they are very few in number, and that for the most part even these are starved, lacking adequate financial

support, adequate selection of students and adequate staff. Again the old obstacle interferes: it still seems impossible for the community at large to treat this form of education seriously.

Before closing this discussion of nursing education and nursing schools there is something yet to be said, and all who have followed recent events in this field will understand the reason for adding this. I hope strongly that there will be no class distinction between the graduates of the university school and those from the junior or nonuniversity school. Surely all graduate nurses can be members of the same professional association. Let us put no arbitrary restriction upon the word *professional*. I should add one further word concerning the assistant or auxiliary worker in connection with nursing service. She is important, and her preparation is important, but I shall leave the subject there, for it is quite outside the scope of my present topic.

In conclusion, I must rest content if I have provoked some thought in response to the questions raised at the beginning of this paper. It is evident that in this half-century nursing has been established in far-reaching proportions, and that recruits of reasonably good caliber are now available in large numbers. So far, so good. But there is a less satisfactory aspect of my story. I have claimed that much more thought should be given to the conditions under which hospital nursing is operating, and, above all, that the educational resources of nursing need intensive development. I hope that the total description merits the title of progress; and I hope also that the next fifty years will enable nursing to build well upon the foundations that have been laid between 1900 and 1950.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 37121

PRESENTATION OF CASE

A thirty-six-year-old woman was admitted to the hospital because of sterility.

The patient stated that fifteen years before entry she had had gonorrhea, associated with a great deal of vaginal discharge and mild lower-abdominal pain. This cleared up under treatment. The patient had been married for eleven and a half years and had been unable to become pregnant. A Rubin test was performed about two years before admission; no gas escaped through the tubes. Later that month a uterotubogram revealed a normal cervical canal and uterine cavity. No oil entered the right fallopian tube. The left fallopian tube filled, with the formation of a cluster of iodized oil just below the visualized end of the tube. Films taken twenty-four hours later showed remnants of oil in the midline apparently in the vagina and uterus and the pool of oil still present in the left pelvis (Fig. 1). Pelvic examination at that time was normal; however, two months later the patient had a delayed menstrual period and at that time was found to have a small, slightly tender cystic mass in the left vault. An Aschheim–Zondek test was negative. The patient had a menstrual period two weeks later, and over the next six weeks the mass in the left vault disappeared. Aside from this episode the patient's periods had occurred regularly every twenty-eight days with a three-to-five-day moderate flow and no associated cramps. Examination of the husband revealed that he was fertile.

The family history was noncontributory. The past history revealed that the patient had not had any other serious illnesses and had undergone no operations.

Physical examination showed a well developed and well nourished woman. The chest was clear. The heart sounds were of good quality, with no murmurs. The uterus was in the anterior position. There was tenderness in both vaults; no masses could be palpated bimanually.

The temperature was 98.6°F., the pulse 74 and the respirations 16. The blood pressure was 90 systolic, 60 diastolic.

Examination of the blood revealed a hemoglobin of 12.4 gm. and a white-cell count of 6900, with a normal differential. The urine was normal.

An operation was performed on the second hospital day. A photograph of the operative findings is submitted (Fig. 2).

Differential Diagnosis

Dr. Arthur Hertig*: This is a rather novel approach to a clinicopathological conference, but I should like first to discuss the case on the basis of the history before I show the photograph of the structure exposed at operation. On the basis of the history, it is quite possible that this delayed period followed by a normal one some two months after she was investigated for sterility by a uterotubogram may well have been due to a "chronic" ectopic pregnancy. Such a pregnancy is one that

Figure 1.

does not rupture but slowly involutes over a period of weeks or months. I have seen such cases with a clinical history of some months or even of years. It is possible, but I do not believe this condition to be a "chronic" ectopic pregnancy. I have seen a patient on whom insufflation was done, on the basis of the history and the menstrual cycle, at about the time of the transportation of the ovum through the interstitial portion of the tube. That patient subsequently developed a cornual pregnancy. In spite of such potential dangers it is the policy of some physicians dealing with a sterile patient to do repeated gas insufflations and uterotubograms in the hope that it will enable the patient to become pregnant by keeping the tubes clear.

The condition of the right fallopian tube at operation could be in some way related to the instillation

Figure 2.

of lipiodol into the genital tract. I know that this patient had gonorrhea in the preantibiotic days, so that it is possible that at the time the lipiodol was put in she had a blocked tube, at least on the right — although there seems to be x-ray evidence that the left tube was open to the oil. Possibly some oil flowed into the right tube in amounts insufficient to be seen in an x-ray film. On carbon-dioxide insufflation, however, there was no passage of gas, even though the lipiodol did enter the left tube.

That brings up the possibility that this whole clinical history may well have been due merely to a healing follicular salpingitis. I am inclined to believe that the ultimate pathological diagnosis was a healing follicular salpingitis, probably of gonorrheal origin, with a superimposed foreign-body reaction, a lipiodol granuloma, if you will, or a granulomatous response to the lipiodol that was injected. We know that lipiodol may be present in the tubes and in the pelvis for some years after instillation. It is a curious coincidence that only this morning I saw in the Free Hospital for Women a lipiodol granulomatosis on the surface of an ovary in a patient who had sterility presumably caused by endometriosis. From merely looking at the tissue it could be deduced that the patient had had lipiodol instillation. We called the clinician and were told that she had had an instillation of lipiodol about four years prior to the operation. The gross specimen looked not unlike the picture presented with this case. I should like to see the x-ray evidence that the oil really filled the tube and flowed into the pelvis, because that brings up a very important point: namely, that the peritoneal reaction may have been due to lipiodol granulomatosis.

Dr. Stanley M. Wyman: I am much interested in the point that Dr. Hertig has brought up — the question whether or not the collection of lipiodol

*Pathologist, Boston Lying-in Hospital and Free Hospital for Women.

seen in the left side of the pelvis actually has escaped into the free peritoneal space. You see on the films a grossly normal-appearing uterus and left tube. The contrast substance pools in droplets on the day of the original examination, and a film taken twenty-four hours later shows it remaining in a very sharply circumscribed area (Fig. 1). I do not believe that it has escaped to the free peritoneal space. I do not know how one could differentiate its being in a cavity at the end of the tube and its being in immediate apposition with the tube.

DR. HERTIG: The only thing in contradiction is that the tube that was shown in the photograph was said to be the right tube. I imagine that the material was in the peritoneal cavity, but there is no proof that it was. It is of interest that the gas did not go through during insufflation.

May we see the photograph of the specimen taken at the time of operation that was presented to me (Fig. 2)? I am supposed to determine the nature of the specimen and what should be given as advice to the surgeon.

DR. FRANCIS INGERSOLL: On the left tube there were little white spots similar to those on the right one.

DR. HERTIG: As I looked at the picture before I had any inkling of the clinical story, the first thing that suggested itself was that the small discrete elevated areas were so-called Walthard's rests or peritoneal inclusion cysts that occur commonly on the surface of the fallopian tube. Most tubes have them, in fact. However, they are not ordinarily this large, numerous or prominent.

The next point is whether or not these were tubercles. I have seen one specimen of tuberculosis that showed structures like this, although in tuberculosis of the pelvis the fallopian tubes are open. This tube obviously had been closed.

Another possibility is tubal carcinoma — a primary papillary adenocarcinoma of the fallopian tube. She was within the age limits between which that disease occurs. Most of the patients are in the fifties or older, but there have been such cases in patients as young as eighteen years of age. They are uniformly associated with inflamed tubes — usually on the basis of gonorrhea — therefore the end of the tube is sealed off in cases of tubal carcinoma.

In summary, I should say that this patient had healing follicular salpingitis, possibly of gonorrheal origin, with a lipiodol or oily granulomatous reaction not only within the lumen of the tube but also on the surface and in the pelvic peritoneum — and that therefore the lipiodol did go through the left tube out into the pelvic peritoneal cavity but some of the lipiodol was probably in the right tube also.

DR. INGERSOLL: You have seen just what I saw at operation, except that Dr. Hertig could not tell from the picture that the left tube also was closed. One point I should like to make is that the x-ray picture is characteristic of a hydrosalpinx in which droplets are formed when the oily matter enters a water medium. When droplets collect at the time one is taking the x-ray film one can almost be sure that there is a hydrosalpinx. We had the additional information that the left tube was not open.

At operation we were faced with the problem of a patient with sterility who had begged me: "Do not remove any of my organs. Do not do anything to me unless you can do something to help me get pregnant. I do not want my uterus taken out or any other organs removed." My initial reaction to what was found was: "This is tuberculosis." We removed the right tube, which was blocked at both the cornu and the fimbriated end, and did a plastic operation on the left tube, opening the fimbriated end. We then anxiously awaited the pathologist's report.

DR. HERTIG: I still think she had gonorrheal salpingitis with a superimposed lipiodol foreign-body reaction. I have even seen one patient who had both tuberculosis and gonorrheal salpingitis. Although it is a distinct possibility in this case, I do not think it was tuberculosis.

CLINICAL DIAGNOSES

Sterility.
?Hydrosalpinx.

DR. HERTIG'S DIAGNOSES

Healing follicular salpingitis, gonorrheal.
Granulomatous reaction to lipiodol, of lumen and surface of tube and of pelvic peritoneum.

ANATOMICAL DIAGNOSES

Walthard's rests on salpinx.
Salpingitis, old, gonorrheal.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: The surgical specimen we received showed very clearly the sealed-off tube and these nodules, which appear to shine a bit in the photograph. It is interesting to mention that when I called Dr. Hertig on the telephone to ask him if he would discuss this case and told him about the nodules he said, "Oh, you mean Walthard's rests." Well, that is what they were. On microscopical examination these nodules proved to be for the most part cystic and lined with single and sometimes three or four layers of cuboidal cells — findings characteristic of Walthard rests. There was no evidence of any inflammatory reaction of the type seen with lipiodol. The wall of the tube showed a marked chronic inflammatory process, that had healed, with a great deal of fibrosis — just what one would expect to find in gonorrheal salpingitis.

DR. HERTIG: Then in defense of my position, I shall say that the tube that was left had lipiodol granulomatosis!

DR. INGERSOLL: There is no doubt that there is a real hazard in the use of oil. In the films of one

patient who had lipiodol, one could not distinguish between tuberculosis and these granulomas. The patient was actually treated with streptomycin. It is a difficult differentiation to make.

DR. CASTLEMAN: Usually it is more difficult to distinguish tuberculosis from carcinomatous implants than from Walthard cysts.

CASE 37122

PRESENTATION OF CASE

A fifty-nine-year-old woman was admitted to the hospital because of chest pain.

For about one year the patient had had upper-left-chest pain variously located in the substernal area, supramammary region, left shoulder and scapula. This was constant and was unrelated to food ingestion or activity. During the last two months she had also had pain radiating down the inner aspect of the left arm into all fingers. The patient had had rather numerous aches during the last few years, including low-back pain, bursitis in the right shoulder and arthritis, and found it difficult to distinguish the present pain from these. Aside from the pain she had felt generally well, although she did have some cough and thought that her voice was hoarse. There had been no sputum or hemoptysis. Her appetite was good; she did not think that she had lost weight. There was no history of food intolerance, but the patient experienced constant heartburn with meals. She had noted constipation and the occasional passage of bright-red blood by rectum. She had had some exertional dyspnea and slight evening ankle edema.

The patient had had an appendectomy and hemorrhoidectomy twenty-five years before entry, and a second hemorrhoidectomy ten years previously. The menopause occurred at the age of fifty-three years.

Physical examination revealed a well developed and well nourished woman in no acute distress. The pupils reacted normally and were regular, but the right pupil was larger than the left. There were a moderate number of small, discrete, movable axillary lymph nodes bilaterally. The lungs were clear, the heart and abdomen negative.

FIGURE 1. *X-ray Films of the Chest. Arrows point to margin of mass.*

The temperature, pulse and respirations were normal. The blood pressure was 115 systolic, 80 diastolic.

The urine had a specific gravity of 1.010 and was otherwise normal. Examination of the blood disclosed a hemoglobin of 13.0 gm. per 100 cc. and a white-cell count of 7000, with 47 per cent neutrophils, 36 per cent lymphocytes, 12 per cent monocytes, 4 per cent eosinophils and 1 per cent baso-

phils. A blood Hinton test was negative. An electrocardiogram was normal.

A roentgenogram of the chest showed, within the posterior basal segment of the left lower lobe, a rounded mass 6 cm. in diameter with a sharply defined periphery, closely applied to the diaphragmatic surface (Fig. 1). This was not identified by fluoroscopy. The density of the mass was homogeneous. The lung fields were otherwise normal. There was lobulation of the right diaphragm; the costophrenic angles were clear. The heart was of normal size.

An operation was performed on the fourth hospital day.

Differential Diagnosis

Dr. Laurence L. Robbins*: The first films of the chest were taken outside about a month and a half before admission. At that time I think the fluoroscopist must have had difficulty in identifying structures, because a week later the patient came back and a spot film was taken with barium in the esophagus and two weeks later more spot films were taken, so somebody else was puzzled also. That gives me some comfort, at least. Among our films is a grid film. I shall try to point out the mass on the films. The mass lies medially in the left lower chest and seems to peel away from the diaphragm, which must mean that it is either attached to it or in close association with it. It could be within the lung; if the mass really were within the lung it would be one that invaded the diaphragm to give this appearance. Secondly, it could be a mass on the diaphragm. Thirdly, it could be a lesion coming through the diaphragm.

What lung lesions should be considered? I think carcinoma is a possibility. There was nothing in the history indicating carcinoma, but that does not exclude it, as we see a large number of silent carcinomas these days. However, there is very little change in the adjacent lung and certainly no visible evidence of extension of tumor from the particular mass.

As far as the benign tumors are concerned, I have to think about hamartoma or bronchogenic cyst, and a large number of others. One thing that is against its being a cyst but does not exclude it entirely is the fact that the mass is somewhat irregular in outline.

*Chief, Department of Radiology, Massachusetts General Hospital.

The lesions of the diaphragm that must be considered are the various tumors. Most of them of the fibrous-tissue group. One thing about which I am not at all certain, and that may be a false observation, is that I have a definite impression of seeing a capsule overlying this mass. This particular sign indicating a fat-containing tumor is relatively common in other parts of the body but I have never seen it in the chest. If a lesion is a fat-containing tumor, one can usually see what seems to be a capsule — or, rather, one sees the inside of the capsule on the film, because the density of the surrounding tissue is different from that of the fat adjacent to it. I cannot help toying with the idea that this could be a fat-containing tumor.

Could it be a mediastinal tumor? That seems rather unlikely, because it does not appear to be related to the mediastinum or to the intercostal nerves. There is no bony erosion that I have been able to see. If this were an aneurysm of the aorta, it would be extremely unusual. This mass lies fairly close to the aorta, however.

The possibility of a lesion that has herniated through the diaphragm is one that cannot be disregarded. One rather interesting finding is that the splenic shadow is considerably smaller than usual; also, the gas bubble of the stomach lies farther forward than usual, and that would leave room enough for the spleen to lie posteriorly — or possibly this is just a small spleen. That worries me considerably! If it were simply a spleen that had herniated through the diaphragm, I do not think Dr. Sweet or Dr. Castleman would be interested in putting me on the spot here, because I think they would expect me to recognize that.

If this mass herniated through the diaphragm it should have gone through fairly close to the so-called foramen of Bochdalek or, possibly, through an unusual rent in the diaphragm. I have seen retroperitoneal tumors extending through this space, or potential space, in other cases; I certainly think that it must be considered a possibility. It could be any other of the normal structures that are found anatomically in the left upper quadrant — the adrenal gland, the tail of the pancreas, the kidney or the spleen. Two structures that may frequently herniate in this location are the colon and the small bowel. The mass is too far back to be the usual herniation of the stomach. The fact that on several examinations we were never able to find any

definite air bubble within the mass makes me doubt that it was bowel.

Where do I stand? Actually, the only thing I have a right to say, from what I see on the x-ray films, is that a mass is seen in the posterior portion of the left chest, medially; from there on, anything I say is pure guesswork. I think one must first consider a lesion arising on the diaphragm or coming through it. I think that a lipoma is something to be considered; beyond that I must think of the possibility of herniation of any other normal structure through the diaphragm. My first choice would be a lipoma arising from or below the diaphragm. The suggestion of a dense shadow marking out what seems to be capsule is consistent with the pleura's being pushed up as fat goes through the foramen of Bochdalek.

Dr. Daniel S. Ellis: What do you think the pain was due to, Dr. Robbins?

Dr. Robbins: There is evidence on the films that she had bursitis, but I think the pain was related to the diaphragmatic lesion.

Clinical Diagnosis

Benign mediastinal tumor, ?neurofibroma.

Dr. Robbins' Diagnosis

Lipoma herniating through foramen of Bochdalek.

Anatomical Diagnosis

Diaphragmatic hernia: omental fat herniating through foramen of Bochdalek.

Pathological Discussion

Dr. Benjamin Castleman: Dr. Sweet operated on this patient. Perhaps he will tell us what was found. No specimen was removed.

Dr. Richard H. Sweet: When I exposed the tumor at operation it was clearly attached to the diaphragm, not to the lung. I had not regarded it clinically as a pulmonary tumor but thought it was probably mediastinal. When I first saw the mass I thought it was a lipoma, because of the glistening fatty substance covered by pleura. However, when I incised the pleura the mass proved to be omental fat herniating through the foramen of Bochdalek — an oval hiatus in the diaphragm exactly in the position of the peculiar defect in the diaphragm noted on x-ray examination. The omental fat apparently herniated from the gastrosplenic ligament. That could account for the unusual position of the spleen.

Dr. Richard Schatzki: How did omentum come to be behind the fundus of the stomach?

Dr. Sweet: I do not think it did; I think the omentum came up in front of the stomach.

Dr. Robbins: We know that omentum, colon and small bowel have all been found in hernias coming through the foramen of Bochdalek.

Dr. Sweet: The position of the foramen may vary immensely; it may be quite far laterally or it may be fairly close to the hiatus. This one was perhaps a few centimeters from the left margin of the esophageal hiatus. That is somewhat farther back than in the average case.

Dr. Walter Bauer: Do you mind telling us what a foramen of Bochdalek is?

Dr. Sweet: It is an aperture that exists due to the lack of fusion of some of the embryonic components of the diaphragm. It occurs in the peripheral portion, away from the esophageal hiatus.

Dr. Robbins: Would you say that there is usually no peritoneal covering on it?

Dr. Sweet: That is correct. As a rule, there is merely a hole in the diaphragm. It never has a peritoneal covering. Some of the patients I have operated on have had small bowel or colon in the thoracic cavity. When the bowel was pushed back, there was only an opening, with neither pleural nor peritoneal covering.

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PROGRESS IN CHEMOTHERAPY OF TUBERCULOSIS

THE Tenth Conference on the Chemotherapy of Tuberculosis was held in Atlanta, Georgia, January 25-28, 1951. Participating in this conference were representatives of 42 study units of the Veterans Administration, Army and Navy Hospitals and many investigators and consultants from other institutions. The deliberations and the practical conclusions reached in this conference, like those of other conferences, are another landmark in the progress of the treatment of tuberculosis.

The discussions at this conference were based on observations made chiefly in the various study groups. From these groups data have been collected and analyzed on what is perhaps the largest number of carefully studied and controlled cases of tuberculosis in history. More than 6000 such cases have been studied in their original course of treatment, and over 2000 have been observed in courses of retreatment. Thorough follow-up studies have been made in the majority of these cases.

Significant is the change of title from that of the earlier meetings, which were called Streptomycin Conferences. This is in keeping with the activities of the collaborating units, which are now engaged in the evaluation of various chemotherapeutic and antibiotic agents that give promise of improving the treatment of tuberculosis, either alone or in conjunction with other established methods of medical and surgical management. Thus, in addition to discussions of streptomycin, progress reports were heard at this conference on various aspects of the use of para-aminosalicylic acid (PAS), thiosemicarbazone (Tebione, TB1), neomycin, terramycin and viomycin.

The detailed report of the deliberations of this conference will be published in its transactions, but it is hoped that, as on previous occasions, those conclusions that are of general interest will be summarized and made available for all practicing physicians. A number of interesting and important original papers on the clinical and laboratory aspects of tuberculosis and on its chemotherapy were presented at this conference. These papers will also appear in the transactions; most, if not all, of them will probably be published in scientific journals and thus be made generally available. Meanwhile, some of the practical highlights might be mentioned.

Progress in the treatment of such a chronic disease is necessarily slow, and any new agent that seems promising must undergo careful scrutiny for effectiveness and toxicity before it can be accepted as useful. Controlled observations in a large number of patients over a long period must be made before the new drugs can properly take their place in the armamentarium of the phthisiologists. These principles have guided the members of the conference in making their decisions.

It was the consensus of the participants at this conference that at present there is still no chemotherapeutic or antibiotic agent that of itself approaches streptomycin as therapy for tuberculosis.

Of the agents tested, it was generally agreed that PAS is the most useful. However, this agent by itself has been found to have limited beneficial effects; its major value is as an adjunct to streptomycin therapy. The use of PAS in conjunction with streptomycin, however, has permitted extensions of chemotherapy to additional groups of patients previously considered unsuitable for treatment. PAS has also prolonged the period of effective streptomycin therapy by delaying the emergence of streptomycin-resistant variants of tubercle bacilli. In addition, the combined treatment may also permit reduction in the dosage of streptomycin and thereby diminish to some extent the serious toxic effect of prolonged use of that agent.

Some practical points concerning improvement in therapy were therefore agreed upon by the conferees. It was decided, first of all, that effective initial therapy for tuberculosis should include full doses of PAS (averaging 12 grams daily for adults) in addition to streptomycin. Longer periods of treatment were advised, as compared with the regimens previously practiced or recommended. When used in conjunction with daily doses of PAS, the doses of streptomycin could probably be reduced to 1 gram every three days or 1 gram twice weekly without appreciably reducing the efficacy of treatment, as compared with the benefit of similar doses of streptomycin given daily. Any reduction in efficacy may be compensated for by the delay in emergence of streptomycin-resistant strains of tubercle bacilli, thus permitting effective therapy over longer periods. The combined streptomycin-PAS therapy also offers the possibility of beneficial effect in patients requiring further treatment after the emergence of streptomycin-resistant variants; in such cases the combined treatment appears to be more effective than PAS alone.

Some observations concerning the use of cortisone and ACTH on tuberculous infections in man and animals were also presented at the conference. Much of the evidence indicated that tuberculous infections may be aggravated, under certain conditions, during treatment with these hormones. It was therefore suggested that physicians should be urged to exercise great caution in the use of ACTH or cortisone in patients who might have active or latent tuberculosis.

The director and staff of the Tuberculosis Division of the Veterans Administration and the workers in the participating study units are to be congratulated on the high caliber of their performance as demonstrated at this conference. For the participants, many of whom work in hospitals remote from the great medical centers, this was most stimulating and informative. All this will accrue to the benefit of the patients under their care.

This function of the medical department of the Veterans Administration, together with the affiliation of increasing numbers of the veterans' hospitals with medical schools and the selection of their staffs through deans' committees, has served to give the veterans of the country the highest quality of medical service available anywhere. It is hoped that all these activities will continue to function effectively in the difficult times ahead. It is also hoped that these activities can be kept free from political controls and pressures, so that the great progress made by these hospitals in recent years may continue unabated under its new administrator.

PRACTICE OF PHYSICAL THERAPY

A DEGREE of confusion has been occasioned in the Commonwealth by the existence of two organizations concerned with physical therapy. The Society of Graduate Physical Therapists, which might politely be said to represent the goats, is allegedly made up of the graduates of substandard and commercial schools of physical therapy. The Massachusetts Chapter of the American Physical Therapy Association, on the other hand, which may with equal propriety be considered to represent the sheep, is composed of the graduates of those schools approved by the Council on Medical Education and Hospitals of the American Medical Association.

Last year a bill was sponsored in the legislature that provided for the regulation of the practice of physical therapy in the State. Not only was this

a mandatory bill, which prohibited visiting nurses, licensed attendants and workers in physicians' offices from carrying out any form of physical therapy whatever, but also it failed to provide at all for medical direction. The bill was defeated, although its passage had been favored by the legislative committee on public health.

A bill has been filed in 1951 by Mr. Charles J. Dunn, counsel for the Committee on Legislation of the Massachusetts Medical Society, at the instance of the Massachusetts Chapter of the American Physical Therapy Association. This bill defines a physical therapist as "a person who practices physical therapy as defined in this act under the prescription, supervision and direction of a person licensed in this state to practice medicine and surgery." The use of roentgen rays and radium for therapeutic purposes is excluded in the definition of physical therapy.

The passage of this bill should be encouraged.

RING OUT, WILD BELLS

WEDDING bells rang more furiously in the United States in 1950 than in the previous year, according to a release from the Metropolitan Life Insurance Company, with the celebration of nearly 1,675,000 nuptials. This 5 per cent increase in trips to the altar, which still fell short of a stampede, was apparently occasioned by the situation in Korea, for the first half of 1950 showed a rate below that of 1949. Rifles have apparently been more effective in expediting the state of wedlock than even the traditional shotgun.

It appears that the young men of the country, as if in stubborn opposition to the policies adopted in the totalitarian strongholds of the world, have been increasingly loath to lead their brides down the aisle since the first phase of World War II came to its close in 1945. The rate of 7.7 marriages per thousand population in March, 1950, was, in fact, one of the lowest in the past twelve years.

The largest increase in happy endings took place in the Mountain States; the greatest drop below 1949 levels, inexplicably, occurred in Kentucky.

The statisticians of the Metropolitan Life Insurance Company, although working feverishly on the problem, have little hope to offer. The present outlook is that the marriage rate in the predictable future will drop to one of the lowest levels in the history of the country.

In the days when wars were fashionable, numerous were the hairbreadth 'scapes recorded by writers on military surgery, some of them, indeed, taxing our credulity to an almost unjustifiable extent.

Boston M. & S. J., *March 26, 1851*

MASSACHUSETTS MEDICAL SOCIETY

TREASURER'S REPORT COVERING REFUND DISTRIBUTION

The Treasurer of the Massachusetts Medical Society makes the following report regarding the refund to the district societies for 1951.

The Finance Committee voted to distribute the sum of $4000 to the district societies. The total number of payments of annual dues received by the Treasurer by March 1, to be counted for the refund, was 4124. Therefore, the refund to the district societies for each paid fellow is 0.97.

The following table gives the number of payments, as of March 1, and the refund to each district as of March 7.

DISTRICT	NUMBER REPORTED PAID	REFUND
Barnstable	35	33.94
Berkshire	122	118.33
Bristol North	59	57.22
Bristol South	180	174.59
Essex North	149	144.52
Essex South	230	223.09
Franklin	32	31.03
Hampden	268	259.93
Hampshire	50	48.49
Middlesex East	117	113.48
Middlesex North	94	91.17
Middlesex South	830	805.07
Norfolk	761	738.14
Norfolk South	124	120.27
Plymouth	130	126.09
Suffolk	530	514.07
Worcester	331	321.04
Worcester North	82	79.53
	4124	$4000.00

ELIOT HUBBARD, JR., M.D., *Treasurer*

APPLICANTS FOR FELLOWSHIP

PUBLISHED IN ACCORDANCE WITH THE PROVISIONS OF CHAPTER V, SECTION 2, OF THE BY-LAWS

BARNSTABLE

BEAM, FORREST, JR., Hyannis Road, Barnstable.
Emory University School of Medicine, 1946.

MURAD, FRANZ RENE, Main Street, East Brewster.
Vienna University, 1928. Sponsor: Edward F. Briggs, Truro.

RICE, JACKSON MAURICE, 394 South Street, Hyannis.
Middlesex University School of Medicine, 1931. Sponsor: Paul P. Henson, 149 Main Street, Hyannis.

Sheldon L. Hunt, *Secretary*
Grand Army Highway, Yarmouth Port

BRISTOL NORTH

GAMBILL, JOHN RANDOLPH, JR., Taunton State Hospital.
University of Louisville School of Medicine, 1946.

LINDBERG, OLIVER S., Fuller Memorial Sanitarium, So. Attleboro.
College of Medical Evangelists, Los Angeles, California, 1923.

WILK, EDWARD KENNARD, Box 151, Taunton.
Baylor University College of Medicine, Houston, Texas, 1936.

James A. Bryer, Jr., *Secretary*
170 North Washington Street, No. Attleboro

BRISTOL SOUTH

ARMINIO, JOHN A., 1149 So. Main Street, Fall River.
Middlesex University School of Medicine, 1937. Sponsor: Francis J. Petrone, 140 Winter Street, Fall River.

James E. Fell, *Secretary*
181 Purchase Street, Fall River

ESSEX NORTH

McNALLY, JOSEPH THOMAS, 56 Lexington Street, Lawrence.
Georgetown University School of Medicine, 1945.

Harold R. Kurth, *Secretary*
57 Jackson Street, Lawrence

ESSEX SOUTH

BLACKBURN, NELSON PHILLIPS, 543 Lynnfield Street, Lynn.
Columbia University College of Physicians and Surgeons, 1945.

COLLINS, WILLIAM JAMES, 25 Conant Street, Danvers.
Tufts College Medical School, 1942.

FILIP, B. JOHN, 416 Essex Street, Salem.
Kansas City College of Medicine and Surgery, 1933. Sponsor: Alexander Kotarski, 109 Main Street, Peabody.

LUNDBERG, ROBERT NELSON, 1037 Washington Street, Gloucester.
Cornell University Medical College, 1944.

MARMER, GEORGE, 66 Lewis Street, Lynn.
Middlesex University School of Medicine, 1941. Sponsor: S. E. Steiman, 23 Broad Street, Lynn.

STOREY, CHARLES MOORFIELD, JR., Northern Avenue, Essex.
Harvard Medical School, 1943.

WELLS, EDWARD PHILLIPS, Wing Road, Lynnfield Center.
New York University College of Medicine, 1942.

Henry D. Stebbins, *Secretary*
342 Essex Street, Salem

HAMPDEN

BALDWIN, JOSEPH VINCENT, North Wilbraham.
Yale University School of Medicine, 1940.

CALLOMON, E. ROBERT P., 296 Hillside Avenue, Holyoke.
University of Cologne, Germany, 1933. Sponsor: Elmer J. Harrington, 199 Chestnut Street, Holyoke.

CREED, JOSEPH CHARLES, 84 Englewood Road, Longmeadow.
University of Vermont College of Medicine, 1944.

EMERY, ROBERT WILLIAM, 3 Providence Street, Springfield.
Tufts College Medical School, 1946.

FERRIS, EMIL MICHAEL, 204 School Street, Chicopee.
Tufts College Medical School, 1946.

GURLAND, HARRY BERNARD, 447 Sumner Avenue, Springfield.
Middlesex University School of Medicine, 1945. Sponsor: Aurel Lavoie, 14 Chestnut Street, Springfield.

MALONEY, JOHN MARTIN, 521 Liberty Street, Springfield.
Georgetown University School of Medicine, 1945.

NERO, WALTER JOSEPH JOHN, 87 Longhill Street, Springfield.
Yale University School of Medicine, 1943.

NORCROSS, PLINY, 15 Pine Street, West Springfield.
University of Illinois College of Medicine, 1940.

Allen G. Rice, *Secretary*
33 School Street, Springfield

HAMPSHIRE

GULBEN, ELVIRA ALEXANDER (GODING), Route 3, Amherst.
Woman's Medical College of Pennsylvania, 1947.

KELLER, JACOB A., Chesterfield.
Columbia University College of Physicians and Surgeons, 1905.

F. Mary P. Snook, *Secretary*
Northampton State Hospital, Northampton

MIDDLESEX EAST

DESFORGES, JANE FAY, 49 Lake Avenue, Melrose.
Tufts College Medical School, 1945.

GOLDMAN, JOSEPH SIMON, 795 Main Street, Wakefield.
Middlesex University School of Medicine, 1945. Sponsor: Howard A. Bouvé, 22 Yale Avenue, Wakefield.

GUARENTE, AMOROSO JAMES, 34 Water Street, Winchester.
Middlesex University School of Medicine, 1942. Sponsor: A. L. Maietta, 408 Main Street, Winchester.

KANE, CHARLES FAIRWEATHER, 41 Hillcrest Avenue, Melrose.
University of Pennsylvania School of Medicine, 1945.

MAHONEY, JOHN PAUL, 7 Tremont Street, Stoneham.
Middlesex University School of Medicine, 1944. Sponsor: Thomas P. Devlin, 34 Pleasant Street, Stoneham.

O'CONNOR, JAMES HENRY, JR., 37 White Street, Winchester.
Tufts College Medical School, 1948.

WINCHELL, GORDON DU FOUR, 256 Woburn Street, Lexington.
Tufts College Medical School, 1944.

William F. Hickey, Jr., *Secretary*
15 Dix Street, Winchester

MIDDLESEX NORTH

JENKINSON, WILLIAM ELLIS, 35 Montview Road, Chelmsford.
Indiana University School of Medicine, 1926.

Philip G. Berman, *Secretary*
174 Central Street, Lowell

MIDDLESEX SOUTH

ANDRUS, STEPHEN BOURNE, 40 Warren Street, Newton Centre.
Johns Hopkins University School of Medicine, 1944.

BALDASSARRE, JOSEPH ANTHONY, 42 Main Street, Somerville.
Kansas City University of Physicians and Surgeons, 1943. Sponsor: George N. Arbeene, 169 Highland Avenue, Somerville.

BELL, DANIEL JACOB, 34 Sunset Road, Cambridge.
Kansas City University of Physicians and Surgeons, 1943. Sponsor: James H. Townsend, 330 Mt. Auburn Street, Cambridge.

BLACHER, SAMUEL, 61 Royal Street, Allston.
Middlesex University School of Medicine, 1943. Sponsor: E. S. M. Landy, 101 Salem Street, Medford.

BROWNSTEIN, NATHAN, 232 Foster Street, Brighton.
Middlesex University School of Medicine, 1941.
Sponsor: Anthony Ferrante, 1242 Commonwealth Avenue, Allston.

CALIVA, FRANCIS SALVATORE, 871 Salem Street, Malden.
Midwest Medical College, Kansas City, Missouri, 1934.
Sponsor: D. Joseph Duggan, 3 Hawthorne Street, Malden.

CARTER, FRANKLIN, 15 Bellevue Avenue, Cambridge.
Harvard Medical School, 1941.

CARTER, GEORGE HERBERT, 26 Farrar Street, Cambridge.
Harvard Medical School, 1943.

CLARK, WILLIAM STRATTON, 46 Gleason Road, Lexington.
St. Louis University School of Medicine, 1938.

COOLIDGE, JOHN COIT, 12 Orchard Street, Belmont.
Yale University School of Medicine, 1944.

DELEROZON, ADRIAN, 372 Lawrence Road, Medford.
Middlesex University School of Medicine, 1939. Sponsor: Charles Butters, 372 Lawrence Street, Medford.

DRISCOLL, JOHN JOSEPH, 300 Grove Street, Melrose.
Tufts College Medical School, 1944.

FLINT, LLOYD DONNE, 144 Newtonville Avenue, Newton.
Yale University School of Medicine, 1941.

FRANKLIN, WILLIAM, 117 Spiers Road, Newton Centre.
Boston University School of Medicine, 1946.

GINSBERG, ABRAHAM, 30 High Street, Everett.
Kansas City University of Physicians and Surgeons, 1937.
Sponsor: Arthur M. Jackson, 512 Broadway, Everett.

KOWALSKI, MARTHA GRAY WILSON, 17 Malvern Terrace, Auburndale.
University of Michigan Medical School, 1944.

LIENWAND, LEO, 1218 Commonwealth Avenue, Allston.
College of Physicians and Surgeons, Boston, 1936. Sponsor: Aaron Feldman, 520 Beacon Street, Boston.

MARCH, ALFRED LÉONARD, 205 Cross Street, Malden.
Middlesex University School of Medicine, 1943. Sponsor: Abraham Bloom, 217 Cross Street, Malden.

McMANAMA, GEORGE BENSON, 59 Hillcrest Road, Belmont.
Tufts College Medical School, 1944.

MORGAN, CHARLES JOSEPH, 83 Main Street, Ashland.
Kansas City University of Physicians and Surgeons, 1943.
Sponsor: Joseph C. Merriam, 198 Union Avenue, Framingham.

PHILLIPS, ROBERT MILTON, 524 Cambridge Street, Cambridge.
Middlesex University School of Medicine, 1945. Sponsor: Myer Marlin, 385 Broadway, Cambridge.

PICCERI, SAVERIO, 104 Mt. Auburn Street, Watertown.
Middlesex University School of Medicine, 1940. Sponsor: Charles Gettes, 247 Mt. Auburn Street, Watertown.

POTTER, HOWARD JOHN, 7 Stetson Street, Lexington.
Boston University School of Medicine, 1945.

ROSENTHALL, CHARLES EDWARD, 179 Elm Street, Somerville.
Middlesex University School of Medicine, 1942. Sponsor: William R. Supple, 400 Mt. Auburn Street, Cambridge.

SANDER, LOUIS WILSON, 20 Forest Street, Cambridge.
University of California Medical School, 1942.

SCINTA, LOUIS ANTHONY, 12 Lincoln Street, Framingham.
College of Physicians and Surgeons, Boston, 1946. Sponsor: James J. O'Leary, 97 Lincoln Street, Framingham.

SELBY, JOHN HORACE, 6 Hemlock Road, Newton Upper Falls.
Boston University School of Medicine, 1944.

SNEGIREFF, LEONID SERGIUS, 72 Radcliffe Road, Belmont.
Syracuse University College of Medicine, 1934.

STREIM, BENJAMIN, 320 Main Street, Watertown.
Kansas City University of Physicians and Surgeons, 1943.
Sponsor: Hyman Shrier, 246 Pearl Street, Newton.

SWAJIAN, ELI H., 812 Moody Street, Waltham.
Middlesex University School of Medicine, 1943. Sponsor: Joseph A. Holmes, 751 Main Street, Waltham.

WALKER, JAMES H., 117 Watson Road, Belmont.
University of Maryland School of Medicine, 1941.

WEEKES, DON JAMES, 67 Marlboro Street, Newton.
Duke University School of Medicine, 1941.

WHALEN, EDWARD JOSEPH, 145 Washington Avenue, West Newton.
Tufts College Medical School, 1946.

WOOL, MAX LEWIS, 3 Elmwood Park, Newtonville.
University of Cincinnati College of Medicine, 1944.

WORONOFF, MORRIS, 1340 Commonwealth Avenue, Allston.
Middlesex University School of Medicine, 1943. Sponsor: James J. Lawlor, 374 Cambridge Street, Cambridge.

ZEITLER, SIDNEY, 108 Salem Street, Malden.
Middlesex University School of Medicine, 1945. Sponsor: Louis Silver, 250 Salem Street, Malden.

ZOLLA, RALPH PASQUALE, 641 Broadway, Everett.
College of Physicians and Surgeons, Boston, 1943. Sponsor: Herbert K. Bloom, 6 Hampshire Street, Everett.

Alexander A. Levi, *Secretary*
481 Beacon Street, Boston

NORFOLK

BARR, JAMES HOWARD, JR., 76 Sherman Road, West Roxbury.
Western Reserve University School of Medicine, 1943.

BOTTOMLEY, GEORGE TAYLOR, 9 Woodman Road, Chestnut Hill.
Tufts College Medical School, 1943.

BOTTOMY, JOHN REUBEN, 62 Sherman Road, Chestnut Hill.
University of Rochester School of Medicine and Dentistry, 1945.

COLBURN, CHARLES GOODSPEED, 272 Allston Street, Brookline.
Boston University School of Medicine, 1945.

DUBROW, AARON ARTHUR, 70 Brown Avenue, Roslindale.
Middlesex University School of Medicine, 1939. Sponsor: Eli Friedman, 416 Marlboro Street, Boston.

FINER, ELLIOT, 290 Fuller Street, Dorchester.
Middlesex University School of Medicine, 1945. Sponsor: John H. Crandon, 520 Commonwealth Avenue, Boston.

GLODT, MILTON, 29 Ober Street, Beverly.
University of Basel, Switzerland, 1936. Sponsor: Samuel Proger, 171 Harrison Avenue, Boston.

GORE, MAX, 217 South Street, Chestnut Hill.
Midwest Medical College, Kansas City, Missouri, 1937.
Sponsor: Jacob Applebaum, 371 Commonwealth Avenue, Boston.

GRYBOSKI, JOSEPH STEVEN, 29 Dorset Street, Dorchester.
Tufts College Medical School, 1945.

HUME, DAVID MILFORD, 112 Washington Avenue, Needham.
University of Chicago School of Medicine, 1943.

GURRI, JOSE NORBERTO, 591 Morton Street, Dorchester.
Havana University Medical School, 1941. Sponsor: Walter E. Barton, 591 Morton Street, Dorchester.

KLEINER, SAMUEL S., 1763 Beacon Street, Brookline.
Zurich, Switzerland, 1948. Sponsor: Jacob Applebaum, 371 Commonwealth Avenue, Boston.

McCARTHY, PHILIP LOUIS, 111 Blue Hills Parkway, Milton.
Tufts College Medical School, 1947.

NESTO, RIBELLE WILLIAM, 34 Fairbanks Road, Milton.
Middlesex University School of Medicine, 1944. Sponsor: Cyril Lydon, 276 Bowdoin Street, Dorchester.

O'CONNOR, JOSEPH JOHN, 172 Church Street, West Roxbury.
Tufts College Medical School, 1947.

ORME, ROBERT JOSEPH, 453 Clapboardtree Street, Westwood.
Boston University School of Medicine, 1945.

PAPPER, SOLOMON, 299 Weston Road, Wellesley.
New York University College of Medicine, 1944.

RODMAN, MELVIN HERBERT, 1 Dunning Way, Jamaica Plain.
Boston University School of Medicine, 1945.

RUBIN, MAURICE WILLIAM, 159 South Street, Jamaica Plain.
Middlesex University School of Medicine, 1942. Sponsor: S. Marcus, 520 Beacon Street, Boston.

SACKS, SEYMOUR, Beth Israel Hospital, Boston.
University of Maryland School of Medicine and College of Physicians and Surgeons, 1943.

THOMPSON, JESSE ELDON, 234 Marked Tree Road, Needham.
Harvard Medical School, 1943.

WEINER, ALBERT E., 4 Griggs Road, Brookline.
Harvard Medical School, 1942.

Basil E. Barton, *Secretary*
10 Richwood Street, West Roxbury

NORFOLK SOUTH

ARNOLD, GORDON DAVIES, 152 Everett Street, Wollaston.
Boston University School of Medicine, 1943.

CONNOLLY, EDWARD PAUL, 520 Summer Street, Weymouth.
Tufts College Medical School, 1945.

LEE, PHILIP HUBBARD, 158 Pleasant Street, So. Weymouth.
Middlesex University School of Medicine, 1932. Sponsor: Robert R. Ryan, 32 Union Street, So. Weymouth.

PATTAVINA, VINCENT, 91 Elm Street, Quincy.
Syracuse University College of Medicine, 1949.

SLOANE, WILLIAM CHARLES, 15 So. Main Street, Randolph.
Middlesex University School of Medicine, 1933. Sponsor: Clifford F. Danforth, 165 Washington Street, Weymouth.

SMITH, ELEANOR F., 70 Tremont Street, So. Braintree.
Tufts College Medical School, 1949.

Ebenezer K. Jenkins, *Secretary*
Norfolk County Hospital, South Braintree

PLYMOUTH

BREIL, NORMAN DAVID, 22 Main Street, Bryantville.
Midwest Medical College, Kansas City, Missouri, 1934. Sponsor: George Moore, Moore Hospital, Brockton.

CALITRI, JOSEPH FRANCIS, 17 Central Square, West Bridgewater.
College of Physicians and Surgeons, Boston, 1942. Sponsor: George Moore, Moore Hospital, Brockton.

GORMLEY, CHARLES JAMES, 4 Cushman Street, Plymouth.
Tufts College Medical School, 1945.

KINCUS, FRANCIS A., 932 North Main Street, Brockton.
Middlesex University School of Medicine, 1943. Sponsor: Edward Farren, 142 Main Street, Brockton.

SHAPIRO, HARRY, 64 Seaver Street, Stoughton.
Middlesex University School of Medicine, 1932. Sponsor: Ralph C. McLeod, 129 W. Elm Street, Brockton.

Samuel Gale, *Secretary*
12 Cottage Street, Brockton

SUFFOLK

BIENVENU, LESTER J., Massachusetts General Hospital, Boston.
Louisiana State University School of Medicine, 1943.

BLOM, GASTON EUGENE, 158 Pine Ridge Road, Waban.
Harvard Medical School, 1944.

BONANNO, SALVATORE VALVO, 51 East Springfield Street, Boston.
Boston University School of Medicine, 1944.

CALLAHAN, EDMUND JAMES, III, 111 Park Drive, Boston.
Harvard Medical School, 1945.

CAMELIO, ALBERT PATRICK, 180 Huntington Avenue, Boston.
College of Physicians and Surgeons, Boston, 1943. Sponsor: Newton C. Browder, 520 Commonwealth Avenue, Boston.

COHEN, JONATHAN, 2 Autumn Street, Boston.
St. Louis University School of Medicine, 1938.

COTTER, THOMAS PAUL, 38 Woodville Street, Roxbury.
Yale University School of Medicine, 1945.

CURTIS, GEORGE WILLIAM, 81 Mount Vernon Street, Boston.
Boston University School of Medicine, 1945.

DE WAN, CHARLES JOSEPH, Boston City Hospital, Boston.
Jefferson Medical College of Philadelphia, 1947.

DUTOIT, CHARLES HILL, 6 Perkins Road, Winchester.
Harvard Medical School, 1942.

FARMER, DOUGLAS ALEXANDER, 382 Warren Street, Needham.
Harvard Medical School, 1942.

FINESINGER, ABRAHAM L., 15 Hereford Street, Boston.
University of Geneva, Switzerland, 1937. Sponsor: Samuel Lowis, Lister Building, Boston.

HIRSCH, ERWIN OSKAR, 106 Sherman Road, Chestnut Hill.
Harvard Medical School, 1946.

IRWIN, JOHN WELLINGTON, 34 Whitfield Road, West Somerville.
University of Pennsylvania School of Medicine, 1942.

KUDARAUSKAS, EDMUND NICODEM, 25 Gardner Street, Allston.
Tufts College Medical School, 1944.

MORRIS, THOMAS ALPHEUS, JR., 232 Beacon Street, Boston.
University of Pennsylvania School of Medicine, 1941.

MUSNICK, HENRY, 7 Grove Street, Boston.
University of Maryland School of Medicine and College of Physicians and Surgeons, 1943.

ROCHLIN, GREGORY, 71 Fresh Pond Lane, Cambridge.
University of Maryland School of Medicine and College of Physicians and Surgeons, 1936.

SEDGWICK, CORNELIUS EDWARD, 11 Shadow Lane, Wellesley.
Cornell University Medical College, 1940.

SIEGEL, JULIUS, 416 Belgrade Avenue, West Roxbury.
Anderson College of Medicine, Glasgow, Scotland. Sponsor: Aaron Feldman, 520 Beacon Street, Boston.

STILES, RICHARD EUGENE, 125 Tyndale Street, Roslindale.
Tufts College Medical School, 1945.

TOCH, RUDOLF, 231 Park Drive, Boston.
Boston University School of Medicine, 1945.

WISEBURG, ISADORE, 204 Proctor Avenue, Revere.
Midwest Medical College, Kansas City, Missouri, 1934. Sponsor: John T. Williams, 429 Beacon Street, Boston.

WOLMAN, HAROLD MELVIN, Metropolitan State Hospital, Waltham.
Boston University School of Medicine, 1942.

Charles G. Shedd, *Secretary*
1180 Beacon Street, Brookline

WORCESTER

EDDY, WARREN HIRAM, 24 Roseland Road, Worcester.
Tufts College Medical School, 1941.

GALE, PHILIP R., Westboro.
New York Medical College, 1946.

JORDAN, HERBERT SMERICAN, 847 Main Street, Worcester.
Kansas City University of Physicians and Surgeons, 1940. Sponsor: Peter A. Colberg, 390 Main Street, Worcester.

ROZANSKI, WILLIAM IGNATIUS, JR., 27 Elm Street, Webster.
Hahnemann Medical College and Hospital of Philadelphia, 1949.

SHANNON, MARY CATHERINE, 334 Highland Street, Worcester.
Kansas City College of Physicians and Surgeons, 1927. Sponsor: Smith Philips, 36 Pleasant Street, Worcester.

SNIFFEN, RONALD CHARLES, 75 Winch Street, Framingham.
University of Toronto Medical School, 1936.

Donald Hight, *Secretary*
57 Cedar Street, Worcester

WORCESTER NORTH

SHACHOY, MARK, 111 Lawrence Street, Fitchburg.
College of Physicians and Surgeons, Boston, 1930. Sponsor: Samuel Golden, 150 Prichard Street, Fitchburg.

SILLS, MALCOLM RICHARD, 20 Prichard Street, Fitchburg.
Middlesex University School of Medicine, 1945. Sponsor: Rudolph Bachmann, 910 Main Street, Fitchburg.

James G. Simmons, *Secretary*
30 Myrtle Avenue, Fitchburg

BOSTON MEDICAL LIBRARY

OSLER AND HIS TEXTBOOK

The Library, in conjunction with Dr. Henry R. Viets, the librarian, has been interested for a number of years in making a collection of the various editions and issues in the different bindings of the famous textbook of medicine written by the late Sir William Osler. The collection now numbers 134 volumes.

First published in 1892, simultaneously in the United States and England, the book went through fifteen editions in America to 1944, and in England through six authorized editions and one pirated edition to 1907. Editions One to Eight were written by Dr. Osler; he was assisted by Dr. Thomas McRae in Edition Nine. Editions Ten to Twelve were revised by Dr. McRae after the death of Dr. Osler, and Thirteen to Fifteen by Dr. Henry A. Christian. The book has been continued by Dr. Christian to date under his own authorship.

The work has been translated into Chinese (three editions), French, German and Spanish.

The first issue is identifiable by the misquotation *Georgias* for the Gorgias of Plato on the reverse of the third page following the title, and in the American issues by the date "November, 1891" at the head of the advertising pages in the back of the volume. The preface by Dr. Osler is dated January 1, 1892. The Plato error also occurs in the first London edition.

It is probably safe to assume that the pages were printed in the United States and a portion sent to London for binding.

The title page for both the American and English editions is the same: *The principles and practice of medicine. Designed for the use of practitioners and students of medicine.* However, the makeup of the title of the London edition is different, and "Fellow of the Royal College of Physicians, London," has been added to Dr. Osler's name. The back strips of the bindings are different, the American binding reading *Appleton's Medical Library. Practice of Medicine. Osler.* The English: *The Principles and Practice of Medicine. Osler.*

A variant of the first issue of the first edition has come into the possession of the Library in which the back strip is lettered in the same fashion as the English edition. It is probable that through an error a few copies were bound in this manner and that this binding constitutes the first issue of the book.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS

The April schedule for Consultation Clinics for Crippled Children in Massachusetts under the provisions of the Social Security Act follows:

Orthopedic Clinics	Date	Clinic Consultant
Salem	April 2	Paul W. Hugenberger
Haverhill	April 4	William T. Green
Greenfield	April 5	Charles L. Sturdevant
Lowell	April 6	Albert H. Brewster
Lynn*	April 9	Daniel M. Killoran; John J. Crowley
Gardner	April 10	Carter R. Rowe
Brockton	April 12	George W. Van Gorder
Springfield	April 17	Garry deN. Hough, Jr.
Worcester	April 20	John W. O'Meara
Fall River	April 23	David S. Grice
Hyannis	April 26	Paul L. Norton

Rheumatic Fever Clinics	Dates
North Reading	April 3, 10, 17, 24
Fitchburg	April 4, 11, 18, 25

Plastic Clinics	Consultant
Eastern Massachusetts	Bradford Cannon
	Semi-monthly
Western Massachusetts	Joseph M. Baker
	Monthly

Physicians referring new patients to clinics should get in touch with the district health officer to make appointments. Patients are seen by appointment only.

*This clinic serves Lynn, Lynnfield, Saugus, Nahant, Revere, Chelsea, Everett and Winthrop. Referrals should be made directly to the Lynn Hospital.

NOTICES

ANNOUNCEMENT

Dr. Jacob I. Weisman announces the removal of his office for the practice of internal medicine to Terhune Hall, 151 Maple Street, Springfield.

HARVARD MEDICAL SCHOOL LECTURE

A lecture will be given in the amphitheater of Building D of the Harvard Medical School on Tuesday, March 27, at 5:15 p.m. Dr. Charles Huggins, professor of surgery, University of Chicago, will speak on the subject "Cancer of the Prostate Gland."

All interested persons are invited to attend.

BOSTON GASTROENTEROLOGICAL SOCIETY

The members of the Boston Gastroenterological Society will be guests at a dinner at 6:30 p.m., Thursday, March 29, given by Captain Nelson in the Marine Hospital, Brighton, Massachusetts. At 8 p.m., after the dinner, Captain Nelson and his staff will present a gastroenterological clinic to which all physicians and medical students are invited.

HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society will be held in the amphitheater of Building D of the Harvard Medical School on Tuesday, April 10, at 8 p.m. (Dr. John R. Pappenheimer presiding).

Program

Cellular Permeability as Affected by Steroid Hormones. D. D. Rutstein and L. M. Leonard, with the technical

assistance of J. H. McIntyre (Department of Preventive Medicine).

The Permeability of the Human Red Blood Cell to Sodium and Potassium. A. K. Solomon (Biophysical Laboratory).

Studies of Potassium Exchange in the Isolated Rat Diaphragm. I. M. Taylor, E. Calkins and A. B. Hastings (Department of Biochemistry).

The Penetration of Cells by Foreign Proteins. A. H. Coons (Department of Bacteriology).

MASSACHUSETTS ACADEMY OF GENERAL PRACTICE

The third annual spring clinical meeting of the Massachusetts Academy of General Practice will be held on April 4.

MORNING SESSION

Amphitheater, Boston University School of Medicine, 80 East Concord Street, Boston

9:00 a.m.–12:00 m. Clinics under the auspices of Massachusetts Memorial Hospitals and Boston University School of Medicine. Program under the direction of Chester S. Keefer, M.D., director, Department of Clinical Research and Preventive Medicine, Robert Dawson Evans Memorial Hospital.

1. Medical clinic.
2. Minor surgery (office procedures).
3. Obstetrics and gynecology.

(The exact content of the program will depend on clinical material available at the time.)

AFTERNOON AND EVENING SESSIONS

Hotel Shelton, 91 Bay State Road, Boston

12:30–1:00 p.m. Registration.

1:00–2:30 p.m. Panel luncheon. Panel speakers will be two or three of those taking part in the morning obstetric and gynecologic clinic.

2:45–3:30 p.m. The Role of the General Practitioner in Civil Defense. Vlado A. Getting, M.D., commissioner of public health, Commonwealth of Massachusetts and head of Medical and Health Services, Massachusetts Civil Defense.

3:30–4:15 p.m. Practical Applications of Adrenal Physiology. Burnham S. Walker, M.D., professor of biochemistry, Boston University School of Medicine.

4:30–6:00 p.m. Business meeting.

6:00–7:00 p.m. Cocktail hour.

7:00 p.m. Dinner.

After dinner Mr. Rudolph Elie, columnist and music critic, *Boston Herald–Traveler*, and roving war correspondent, will speak on the subject "The Roving Eye in Europe."

For further information write to James G. Simmons, M.D., Secretary, 30 Myrtle Avenue, Fitchburg, Massachusetts.

ALUMNI ASSOCIATION, LONG ISLAND COLLEGE OF MEDICINE

The annual alumni day of the Long Island College of Medicine and the State University of New York at New York City College of Medicine will take place on Saturday, April 28. There will be a scientific session at Polhemus Clinic in the morning followed by the 71st annual dinner to be held at the Columbus Club, 1 Prospect Park West, Brooklyn, New York, at 7:00 p.m.

ASSISTANT DIRECTOR FOR CLINICAL RESEARCH

The State of New York Department of Civil Service announces an examination to be held on June 23, 1951, for assistant director for clinical research, Division of Laboratories and Research, Department of Health.

Further information can be obtained from the Examinations Division of the Department of Civil Service, 39 Columbia Street, Albany 7.

NILES MEMORIAL LECTURE

The 10th Annual Walter L. Niles Memorial Lecture will be given at Cornell University Medical College at 8:00 p.m. on Friday, April 20.

Dr. Eugene A. Stead, Jr., will speak on "Congestive Heart Failure." Dr. Stead, who is professor of medicine at Duke University School of Medicine and physician-in-chief at Duke Hospital, Durham, North Carolina, is widely known for his numerous publications on various aspects of cardiovascular disease.

The Walter L. Niles Memorial Lecture, sponsored by the Nu Sigma Nu medical fraternity, is delivered each year at Cornell University Medical College in memory of Dr. Niles, former professor of clinical medicine and dean of the Medical College. In the past the lecture has been delivered by such prominent physicians as Dr. Barry Wood, Dr. William Menninger, Dr. Chester S. Keefer, Dr. Allen Whipple and Dr. Edward Strecker.

Members of the medical profession and others interested in cardiovascular disease are cordially invited to attend this year's lecture.

SOCIETY MEETINGS AND CONFERENCES

MARCH 26. Boston Lying-in Hospital Obstetric Round-Table Conference. Page 629, issue of October 19.

MARCH 26–28. American Academy of Pediatrics. Page 274, issue of February 15.

MARCH 27. Norfolk District Medical Society. Page 629, issue of October 19.

MARCH 27. Harvard Medical School Lecture. Page 457.

MARCH 27 AND 28. Boston City Hospital House Officers' Association. Page 421, issue of March 15.

MARCH 28. International College of Surgeons. Page 422, issue of March 15.

MARCH 28. Naval Reserve Volunteer Medical Unit No. 1-1. Page 384, issue of March 8.

MARCH 29. 94th Infantry Division Lecture. Page 422, issue of March 15.

MARCH 29. Boston Gastroenterological Society. Page 457.

APRIL 4. Massachusetts Academy of General Practice. Notice above.

APRIL 4. New England Pediatric Society. Page 346, issue of March 1.

APRIL 7. Massachusetts Society of Anesthesiologists. Page 346, issue of March 1.

APRIL 7–28. Springfield Hospital. Page 421, issue of March 15.

APRIL 9–13. American College of Physicians. Page 296, issue of June 15.

APRIL 10. New England Society of Anesthesiologists. Page 512, issue of September 28.

APRIL 10. Harvard Medical Society. Page 457.

APRIL 11. Tufts Medical Alumni Association. Page 422, issue of March 15.

APRIL 12. The Diagnosis and Treatment of Kidney Diseases in Childhood. Dr. Charles A. Janeway. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

APRIL 18. Physical Medicine and Rehabilitation in Psychiatry. Dr. Walter E. Barton. New England Society of Physical Medicine. Hotel Kenmore. 8 p.m.

APRIL 20. Niles Memorial Lecture. Notice above.

APRIL 24. Norfolk District Medical Society. Page 629, issue of October 19.

APRIL 28. American Psychosomatic Society. Page 198, issue of February 1.

APRIL 28. Alumni Association, Long Island College of Medicine. Notice above.

MAY 7–11. National League of Nursing Education. Page 384, issue of March 8.

MAY 8. New England Society of Anesthesiologists. Page 512, issue of September 28.

MAY 9. Norfolk District Medical Society. Page 629, issue of October 19.

MAY 16. New England Obstetrical and Gynecological Society. Hillcrest Country Club, Leicester, Massachusetts.

MAY 16–18. American Association of Genito-Urinary Surgeons. Page 160, issue of January 25.

MAY 21–25. International Society of Orthopaedic and Traumatic Surgery. Page 422, issue of March 15.

MAY 22–24. Massachusetts Medical Society. Annual meeting. Hotel Statler, Boston.

MAY 24–26. American Goiter Association. Page 384, issue of March 8.

JUNE 9 AND 10. American Society for the Study of Sterility. Page 274, issue of February 15.

JUNE 11–15. American Medical Association Annual Session. Atlantic City, New Jersey.

JUNE 15–17. American Electroencephalographic Society. Page 346, issue of March 1.

JUNE 20–23. American Orthopaedic Association. Page 422, issue of March 15.

SEPTEMBER 4–8. American Congress of Physical Medicine. Page 238, issue of February 8.

(*Notices concluded on page xi*)